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CLINICAL USE OF THE CHAMBERLAIN TECHNIC IN SACROILIAC CONDITIONS

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AND
VERNON L. PETERSON, M.D.
CHARLESTON, W. VA.

The question of sacroiliac slip has been discussed for years. Snelling published a paper on this subject in 1870. Goldthwaite and Osgood made an important contribution in 1905. Fick, Smith-Petersen and others have also made their contributions. There seems to be no question at the present time but that the sacroiliac joint is a movable joint.

Chamberlain in 1919 first began to use his x-ray technic of demonstrating slipping of the sacroiliac joints. He read a report on this subject at a meeting of the American Roentgen Ray Society in September 1922. Allen published the description of a somewhat similar technic in 1923. Chamberlain [1] has published papers in 1930 and 1932 on this subject.

Chamberlain states that "there is no reason to expect the ordinary anteroposterior view of the sacroiliac region to reveal the normal or abnormal movement occurring in these joints because motion of the sacroiliac joint is not in a direction which could be shown by any sort of x-ray study of the joint itself. The sacroiliac motion, when it occurs, is rotatory, and the rotation takes place within an axis which is perpendicular to the joint surface. The place to look for evidence of sacroiliac joint motion is at the symphysis pubis, where it is magnified and measurable." Chamberlain makes first the ordinary routine stereoscopic posterior views which are made with the patient supine on the Potter-Buckey diaphragm table, and with the symphysis pubis and the entire pelvic rim in the field of view. Also a lateral film is taken, either right or left, centered over the lumbosacral joint.

Next the special technic is then described by him as follows: "Expose the pair of special anterior projections of the pubis, the patient standing with weight bearing on the alternate leg. In these views it is advisable to have the Potter-Buckey diaphragm mounted in the erect position. The patient is standing upon a pair of steps approximately 3 to 6 inches high facing the Potter-Buckey diaphragm. The first film is labeled "right" and the label is placed at the film margin, toward the patient's right, to indicate weight bearing on the right leg. The step is removed from under the left leg and the first exposure is made while the left leg is hanging free. The label is then changed to "left" and is placed at the film margin toward the patient's left. The step is then placed under the left foot, and the one under the right foot is removed. The second film is exposed with weight bearing on the left leg, the right leg hanging free." We take also a third check x-ray film with the patient standing on both feet.

Chamberlain states that "a scale is applied first to one of the weight bearing films and then to the other in such a manner that the vertical line on the scale lies in the soft tissue zone, midway between the bony outlines of the two pubes. The difference in the height of the two sides of the pubes is then measured."

He states that in 90 per cent of his cases the sacroiliac symptoms have been on the side of the high pubic bone. He states that "the normal movements range from nothing to 0.5 mm. in the male and from nothing to 1.5 mm. in the nonpregnant female.

This x-ray technic has been used by one of us, V. L. P., a roentgenologist, and the senior author for the past two and one-half years in 144 cases. A careful history is taken, and the ordinary clinical tests used in low back examinations have been made in all cases. The methods of Nutter, Pitkin [2] and others demonstrating undue mobility of the sacroiliac joint clinically have not been made. The total of 144 cases studied were cases which seemed to be somewhat obscure so far as their diagnosis was concerned. These were consecutive cases and were not selected.

One hundred and seventeen of these patients were male and 27 were female. Sacroiliac slip occurred in 35 of the 117 cases in males, or 29.9 per cent. It occurred in 16 of the 27 cases in females, or 59.2 per cent.

Thirty-five of the 144 cases were private cases and 109 were compensation cases. Twenty-three, or 65.7 per cent, of the private cases showed a slip. Twenty-eight, or 25.7 per cent, of the compensation cases showed slipping. It is believed that this examination is especially useful in compensation cases, as it is well known that they are more difficult to evaluate than ordinary private cases.

The onset of the trouble was due to an accident in 131, or 91 per cent of the whole series of cases, and the onset was gradual in 13, or 9 per cent of the whole series. In the cases showing sacroiliac slip 43, or 84 per cent, were due to accident and 8, or 16 per cent, were gradual in their onset. Thus it seems that while accidents predominate as the cause of slipping it is by no means unusual to see slipping in cases without previous history of accident.

Low back pain was the chief complaint in all of these cases. Associated leg pain was found in 65, or 46 per cent, of the whole series and in 24, or 46 per

1. Chamberlain, W. E.: The X-Ray Examination of the Sacroiliac Joint, Delaware M. J. **4**: 195-201, 1932; The Symphysis Pubis in the Roentgen Examination of the Sacroiliac Joint, Am. J. Roentgenol. **24**: 621-625, 1930.

2. Pitkin, H. C., and Pheasant, H. C.: Sacrarthrogenetic Telalgia: I. A Study of Referred Pain, J. Bone & Joint Surg. **18**: 111-133 (Jan.) 1936.

cent, of the cases showing sacroiliac slip. Then in 54 per cent of the cases leg pain was absent and the pain was confined to the lower back and gluteal region.

A routine back examination was made in these cases with special reference to list, limitation of straight leg raising, limitation of spine motions, Trendelenburg, Gaenslen, Ober and Ely signs, compression and separation of the pelvis, and points of maximum tenderness. The question of posture and limp were all considered.

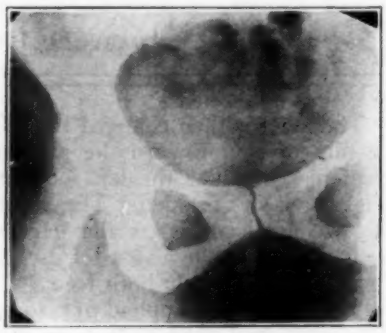

Fig. 1 (patient T.).—Appearance with patient standing on both feet.

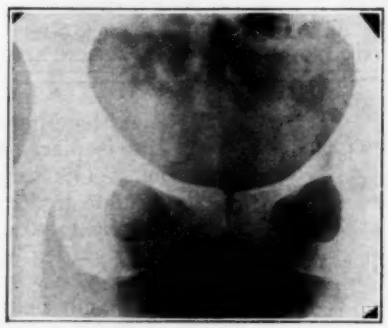

Fig. 2 (patient T.).—Patient standing on the left foot: no displacement in the left sacroiliac joint.

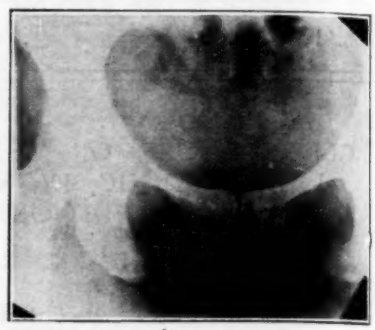

Fig. 3 (patient T.).—Patient standing on right foot: displacement of 2.5 mm.

It is interesting to note that a list was found in comparatively few of these cases.

On the other hand, straight leg raising was found to be limited in 59 per cent of the whole series of 144 cases and was found in 93 per cent of the 51 cases showing sacroiliac slip. Thus it can be said that a normal straight leg raising test will almost eliminate a sacroiliac slip.

As was to be expected, there was considerable limitation of spine motion, that is as regards motion in the lumbar spine, in many cases. This point was of little use in differentiating sacroiliac lesions from other conditions, however.

The Trendelenburg test was positive in comparatively few cases and was found to be of little help.

The Gaenslen test was found to be positive in a little over one fifth of the whole series of 144 cases and also in about the same percentage in the cases showing a sacroiliac slip.

The Ober test was found to be positive in a somewhat higher percentage of the cases. It was positive in 40 per cent of the whole series of 144 cases and in 35 per cent of the cases showing sacroiliac slip.

The Ely test was found to be positive in about one third of the whole series and in only about 25 per cent of the cases showing a sacroiliac slip.

Pain on attempted separation or compression of the pelvis was found to be present in a comparatively small percentage of the cases. It did not seem to be of definite help in making the diagnosis of this condition.

As was to be expected, the sacroiliac joint was the usual point of maximum tenderness. It was present in 76, or 52 per cent, of the whole series of 144 cases and in 30, or 58.8 per cent, of the cases showing sacroiliac slip. Sciatic tenderness was present in less than 1 per cent of the whole series but in 4 cases, or 7 per cent, of the cases showing sacroiliac slip.

All patients showing definite sciatic symptoms were referred to a neurologic surgeon for further neurologic studies and studies with iodized oil if indicated.

Poor posture was found in a comparatively small percentage of these cases showing sacroiliac slip, 3 cases, or 5.8 per cent. A limp was found in only 5 cases, or 10 per cent, of the cases showing sacroiliac slip.

The x-ray examination showed sclerosis in 35, or 24 per cent, of the whole series of 144 cases and this was present in only 8 cases, or 15 per cent, of the cases showing sacroiliac slip.

It has been found by Pitkin and others that sacroiliac slips are present more commonly on the left side than on the right. In our series 15 cases, or 29.4 per cent, showed slipping on the right side and 27 cases, or 52.9 per cent, showed slipping on the left. Nine cases, or 17.6 per cent, had a bilateral slipping by x-ray examination.

Twenty-nine cases, or 20 per cent of the whole series, showed positive sacroiliac signs and the x-ray examination showed a slipping on the same side. Eight cases, or 5.5 per cent of the whole series, showed positive sacroiliac signs on both sides and the x-ray examination showed slipping on both sides. Seventy-one cases, or 49 per cent of the whole series, showed sacroiliac signs which were predominantly negative on both sides and the x-ray examinations were negative on both sides.

Fig. 4 (patient B.).—Appearance with patient standing on both feet.

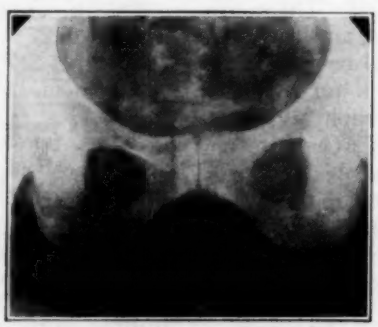

Fig. 5 (patient B.).—Patient standing on left foot with displacement of 1.5 mm.

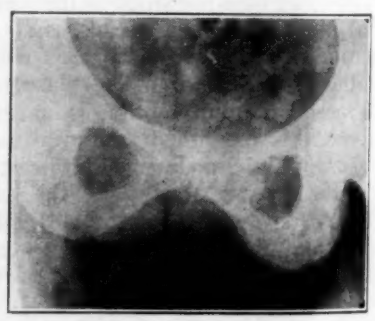

Fig. 6 (patient B.).—Patient standing on right foot, displacement of 3 mm.

Two cases, or 1.3 per cent of the whole series, showed positive sacroiliac signs on both sides and the x-ray examination showed a slipping on one side only. One case, or 0.67 per cent of the whole series, showed positive sacroiliac signs on one side and x-ray examination showed slipping on both sides.

On the other hand, 8 cases, or 5.5 per cent of the whole series, showed sacroiliac signs which were inconclusive and the x-ray examination showed a slipping on the same side. In 6 cases, or 4.16 per cent of the whole series, the sacroiliac signs were inconclusive and the x-ray examination showed a slipping on the opposite side. Also 14 cases, or 9.7 per cent of the whole series, showed sacroiliac signs which were positive on one side and the x-ray examination was negative. Five cases, or 3.4 per cent of the whole series, showed sacroiliac signs which were positive on both sides and x-ray examination were negative bilaterally.

Now, to recapitulate, the sacroiliac signs and x-ray findings agreed completely in 108 cases, or 74.5 per cent of the whole series. The sacroiliac signs and x-ray findings agreed partially in 11 cases, or 7.6 per cent. Thus, in a total of 82.1 per cent the x-ray examination showed agreement with the essential clinical findings. On the other hand, the sacroiliac signs and the x-ray findings were in disagreement in 25 cases, or 17.9 per cent.

The x-ray films were available for review in 42 of the 51 cases showing sacro-iliac slip. Of these cases the right sacroiliac joint was involved in 16 males and 10 females. There was an average of 1.9 mm. of slipping in the male, disregarding the slip in 1 case which was 10 mm. There was an average slipping of 1.8 mm. in the female.

The left sacroiliac joint showed 16 males involved with an average slipping of 1.47 mm., and 11 females with an average slipping of 1.68 mm.

Of the 42 cases involving 53 joints in which x-ray measurements were made, 37 of these cases and 46 joints showed slipping above the normal of 0.5 mm. in the male and of 1.5 or more millimeters in the female. Thirty of these joints were in the male and 16 in the female.

An analysis of these 37 cases is of interest. Twenty-one out of these 37 cases, or 56 per cent, were cases in which the sacroiliac signs were positive and the x-ray examinations showed slipping on the same side. In 5 of these cases, or 13.5 per cent, the sacroiliac signs were positive on both sides and the x-ray examinations showed a slipping on both sides. In a total of 26 out of 37 cases, or 70 per cent, there was complete agreement between the x-ray and the clinical findings. In 2 cases, or 5.4 per cent, the sacroiliac signs were positive on both sides and x-ray examinations showed slipping on one side only. In 2 cases sacroiliac signs were positive on one side and the x-ray examinations showed slipping on both sides. In 5 cases, or 13.5 per cent, the sacroiliac signs were inconclusive and the x-ray examinations showed slipping on the same side. In 2 cases the sacroiliac signs were inconclusive and the x-ray examinations showed slipping on the opposite side.

CONCLUSION

The so-called Chamberlain technic of x-ray examination of the sacroiliac joint is worth while and is accurate in 70 to 80 per cent of the cases. It should be used in doubtful cases of sacroiliac joint conditions.

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FETAL AND NEONATAL MORTALITY IN PREGNANCIES COMPLICATED BY DIABETES MELLITUS

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About 30 per cent of all the pregnancies complicated by diabetes mellitus result in the death of the fetus or the newborn infant. The cause of this high mortality has remained obscure. No significant reduction in fetal deaths has been shown to have occurred since insulin has come into use. Several factors have been held responsible for the high death rate. These include the degree of care with which the maternal diabetes has been regulated, the presence of such complications of pregnancy as toxemia and hydramnios, the frequent occurrence of large fetuses, the increased incidence of congenital anomalies and the hypoglycemia in the liveborn infant. There is no general agreement, however, that any one or that all of these factors together can explain satisfactorily the fetal mortality.[1]

A recent study by Miller and Wilson [2] has brought out the fact that infants born of diabetic mothers may have cardiac hypertrophy and excessive erythropoiesis in the liver with large numbers of nucleated red blood cells in the peripheral blood during the first days of life. The cardiac and hepatic changes were found by them associated sometimes with hyperplasia of the islands of Langerhans in overweight fetuses. The anatomic changes described were noted to have been present in 2 instances before maternal diabetes was manifest. The frequent occurrence of this syndrome in infants born to diabetic mothers suggested that there might be some causal relationship between it and diabetes mellitus. The fact that it was sometimes present before the onset of recognizable signs and symptoms of diabetes indicated that the anatomic changes were not related to

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Many persons contributed information obtained from the records of patients in the three hospitals, especially Dr. Arthur H. Morse and the staff of the Department of Obstetrics and Gynecology, Dr. John P. Peters and the members of the Metabolism Service of the Department of Medicine, Yale University School of Medicine, Dr. Edward Tolstoi of the Department of Medicine and Dr. S. Z. Levine of the Department of Pediatrics, Cornell University Medical College.

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2. Miller, H. C., and Wilson, H. M.: Macrosomia, Cardiac Hypertrophy, Erythroblastosis and Hyperplasia of the Islands of Langerhans in Infants Born to Diabetic Mothers, J. Pediat. **23:** 251, 1943.

the hyperglycemia in the mother. These observations were based on autopsies done on infants at the Boston Lying-in Hospital, the New Haven Hospital and the New York Hospital. The anatomic changes reported by Miller and Wilson were the result of a preliminary survey. A more detailed study of the postmortem material from the three hospitals is to be made in the near future under the direction of Dr. Arthur T. Hertig of the Boston Lying-in Hospital. It was felt that while the autopsy material was being studied a survey of all the pregnancies complicated by diabetes in the three hospitals should be made. The purpose of the investigation was to evaluate all the factors which might have a bearing on the survival of the fetus. Studies of this kind have been made before, but seldom has there been an opportunity to report enough cases to give statistical significance to the results.

The 123 mothers had 143 pregnancies and there were 146 births, the results of which are shown in table 1. The fetal and neonatal mortality in the diabetic pregnancies have been compared to those in 3,079 consecutive births which occurred in the New Haven Hospital from 1928 to 1932 inclusively.[4] The total mortality among the 3,079 births was 5.4 per cent. The mortality among the 146 births to diabetic mothers was 29.4 per cent, or about five times higher than in the nondiabetic group. The fetal mortality was six times and the neonatal mortality was four times higher in the diabetic group than in the non-diabetic. Twenty-two of the 30 fetal deaths were macerated fetuses.

The high death rate among the fetuses and liveborn infants of diabetic mothers is not related primarily to the severity of the maternal diabetes. In the group of 123 diabetic mothers there were 42 who were classified as having diabetes but who did not require insulin at any time during pregnancy. The diagnosis of diabetes was made in these 42 mothers on the fact that they had glycosuria with or without symptoms plus the fact that in all but 2 instances either the dextrose tolerance curve was of the diabetic type or the fasting blood sugar was over 140 mg. per hundred cubic centimeters. There were 48 births in 45 pregnancies among the 42 mothers, and the mortality for the infants is shown in group 3, table 1. The combined fetal and neonatal mortality was 22.9 per cent, or four times higher than the mortality in the nondiabetic group. This high death rate among the offspring of the diabetic mothers who did not require insulin is not the result of maternal complications of pregnancy, for the mortality is almost the same even when the maternal complications are excluded as a possible cause (see Group 3a table 1). Under the most favorable conditions the risk to the infant of a diabetic mother is about four times that to the infant born to a nondiabetic woman. In the presence of severe complications of pregnancy in women

TABLE 1.—*Fetal and Neonatal Deaths in Diabetic and Nondiabetic Pregnancies, Showing Effect of Mild Diabetes and Uncomplicated Pregnancies on the Fetal and Neonatal Mortality.*

Group	Births	Fetal Deaths *		Neonatal Deaths *		Total Deaths		Chi Square
		No.	%	No.	%	No.	%	
1. Nondiabetic	3,079	99	3.2	67	2.1	166	5.4	
2. Total diabetic	146	30	20.5	13	8.9	43	29.4	133
(a) With complications of pregnancy †	55	17	30.9	7	12.7	24	43.6	131
3. Diabetic, not taking insulin	48	7	14.6	4	8.7	11	22.9	27.2
(a) Without complications of pregnancy	34	4	11.7	3	8.8	7	20.6	13.5

* Fetal deaths include macerated fetuses and other stillborn infants. Neonatal refers to the first ten days of life only. These definitions apply in all subsequent tables.

† Complications of pregnancy were as follows: toxemias of pregnancy (all forms), 35; hydramnios, 16; diabetic acidosis, 4; pyelitis, 4; hypertension, 2; syphilis, 1; placenta previa, 1. Some mothers had more than one complication of pregnancy, but they were counted in the table as having only one.

TABLE 2.—*Combined Fetal and Neonatal Mortality Before and After the Onset of Diabetes*

	Diabetic										Nondiabetic			
	Insulin Treated			Not Insulin Treated			Total							
	Born	Died	%	Born	Died	%	Born	Died	%	Chi Square	Born	Died	%	Chi Square
After onset of diabetes	41	11	27.5	52	11	21.1	93	22	23.6					
1-5 years before onset	40	16	40.0	39	12	30.7	79	28	35.4	....	107	8	4.8	43.4
6-10 years before onset	42	6	14.3	37	4	10.8	79	10	12.6	11.1	104	3	2.8	6.5
11-15 years before onset	31	4	15.4	36	4	11.1	67	8	11.9	10.7	79	0	0.0	10.9
16-20 years before onset	10	2	20.0	17	2	11.7	27	4	14.7	4.0	48	1	2.1	4.5

The chi squares in the column marked "Total" represent the significance of the difference between the mortality in the five year period immediately preceding the onset of diabetes and the mortality in each of the other five year periods in the prediabetic era. The chi squares in the column marked "Nondiabetic" refer to the significance of the difference between the mortality in nondiabetic and total diabetic in the corresponding five year periods.

MATERIAL

The material for this study was obtained from the hospital records of all diabetic pregnant women and their offspring admitted to the Boston Lying-in Hospital, the New Haven Hospital and the New York Hospital since 1930, 1928 and 1933 respectively.[3] No diabetic mother was accepted for study who was admitted to a hospital for the delivery of a fetus that weighed less than 1,000 Gm. On this account 5 mothers and their fetuses were excluded. There remained 137 mothers who were observed in the three hospitals through 159 pregnancies. There were 162 births. The statistical significance of the results was determined by the chi square test. A chi square of 3.8 or more may be considered as statistically significant.

RESULTS

In the group of 137 mothers there were 123 whose diabetes had its onset either before or during the pregnancy for which they were admitted to the hospital.

3. Footnote deleted on proof.

4. Hadley, E. E.: Morbidity and Mortality in the Neonatal Period, Thesis, Yale University, 1934.

with diabetes, the risk to the infant may be greatly increased. The mortality among the infants whose mothers had severe complications of pregnancy with both mild and severe diabetes was 43.6 per cent (group 2a, table 1). A clear understanding of the cause of the high mortality in diabetic pregnancies will not be had, however, until the explanation for the high mortality in the noncomplicated group is obtained.

There is ample evidence that the fetal and neonatal mortality is extraordinarily high long before the mothers develop symptoms or signs of diabetes. In table 2 there has been presented the results of all the pregnancies exclusive of abortions and miscarriages that occurred in those mothers who had pregnancies both before and after the onset of diabetes. The diabetic mothers in table 2 have been divided into two groups: those whose diabetes required insulin during pregnancy and those whose diabetes was regulated solely by diet. The appearance of the first sign or symptom as recorded in the history was taken as the onset of diabetes in both groups of women. The births and deaths in the two groups have been given not only for the period when diabetes was present but also for the years before the onset of diabetes. The latter period has been arbitrarily divided into periods of five years each. The births and deaths that occurred in a group of 106 nondiabetic mothers whose average age at the time of the birth of their last infant was the same as that of the diabetic women at the time of onset of their diabetes, 34 years, are also given in table 2. The nondiabetic women were unselected consecutive patients admitted to the New Haven Hospital for delivery in 1943. In both the insulin and noninsulin treated diabetic groups the fetal and neonatal mortality follows the same pattern. The number of deaths increases as the onset of diabetes is approached, and in the five year period immediately preceding the appearance of signs and symptoms of the diabetic syndrome the mortality is higher than in any other preceding period. The statistical significance of the increase in mortality as diabetes approaches is demonstrated by combining the data in the insulin treated and noninsulin treated groups and comparing the mortality in the five year period immediately preceding the onset of symptoms with each five year period before it. Furthermore, the mortality for the diabetic group in each one of the five year periods before the onset of diabetes is statistically significantly higher than the mortality in the corresponding five year period in the nondiabetic group. In the five year period immediately preceding the onset of diabetes the mortality is higher than that after diabetes is recognized, but the difference is not statistically a significant one.

It is well known that babies born to diabetic mothers tend to be overweight. This increased birth weight can be shown to antedate in the offspring the appearance of diabetic symptoms and signs in the mother. In table 3 the incidence of babies who weighed over 5 Kg. at birth is given.[5] In the babies born to mothers before the latter became diabetic a history of a birth weight of 5 Kg. or more was accepted only if the baby had been born in a hospital where accurate weights could be obtained, since histories of home weights are grossly unreliable. The incidence of babies who weighed more than 5 Kg. at birth was 3.9 per cent before diabetes was diagnosed in the mothers and 6.4 per cent after the onset. Statistically, both of these figures are significantly higher than the incidence of similar babies in the nondiabetic population. The earliest recorded birth weight in excess of 5 Kg. among 256 births was ten years before the appearance of diabetic symptoms.

The excessive size of the fetus has been held by some investigators to be responsible for part of the high mortality in infants born to diabetic mothers. The relation between the birth weight and mortality was studied in this series, and the results are given in table 4. There is nothing in the data of table 4 to indicate that an excessive birth weight decreases the infant's chances for survival. It might be supposed that maceration might influence the birth weights adversely in the lower weight brackets, but even with macerated fetuses eliminated the mortality is no more unfavorable for the infants over 4.5 Kg. than those in the normal range. There were 6 infants with birth weights over 5 Kg. and 2 deaths, indicating possibly that in this very high weight range the mortality does begin to increase. The mortality is very high in infants under 2.5 Kg., as would be expected in premature infants.

TABLE 3.—*Incidence of Large Babies in Diabetic Pregnancies Before and After the Onset of Diabetes*

	Total Births	Births Over 5 Kg.		Chi Square
Nondiabetic (Chicago Lying-in Hospital)	20,219	14	0.07	
Diabetic				
(a) Before onset	256	10	3.9	317
(b) After onset	93	6	6.4	382

TABLE 4.—*Combined Fetal and Neonatal Mortality in Diabetic Pregnancies According to Birth Weight*

Birth Weight, Kg.	Births	Deaths	%	Deaths Less Macerated Fetuses *	Macerated Fetuses	%
1.0-2.5	17	12	70.0	5	7	58.3
2.5-3.5	40	11	27.5	7	4	12.1
3.5-4.5	57	12	21.0	5	7	13.4
4.5-6.0	29	5	17.2	2	3	11.1

* Three macerated fetuses were delivered after thirty-two weeks of gestation but were not weighed and hence were not included.

Among the 137 mothers whose records were studied there were 11 who had glycosuria but who could not be considered as having diabetes, as they had either normal fasting blood sugar concentrations or normal dextrose tolerance curves during pregnancy. In 2 instances, cases 6 and 8 of table 5, no tests were done, so that they could not be classified for certain as diabetic and hence were included in this group. In only 1 instance, case 1, did diabetes subsequently develop in a mother. The group of cases in table 5 have been shown because the number of fetal and neonatal deaths, the incidence of large fetuses and the occurrence of cardiac hypertrophy and erythropoiesis in the liver and blood seemed similar to the results observed in the larger group of births among the definitely diabetic mothers shown in the previous tables. There were 4 deaths, in cases 1, 2a, 2b and 3, among the 12 births listed in table 5, or a mortality of 33 per cent. Three of the infants weighed over 5 Kg. at birth, in cases 2b, 3 and 4. In 2 infants an enlarged heart was noted on roentgen examination, cases 1 and 4.

5. The figures for the nondiabetic at the Chicago Lying-in Hospital were obtained from Koff, A. K., and Potter, Edith L., Complications Associated with Excessive Development of Human Fetus, Am.J. Obst. & Gynec. **38**: 412 (Sept.) 1939.

In the same 2 cases large numbers of erythroblasts were observed in the peripheral blood and in another infant, case 3, there were numerous areas of erythropoiesis in the liver at autopsy.

COMMENT

The belief has long been held that the hyperglycemia in the pregnant diabetic woman is responsible for the large number of fetal and neonatal deaths, the excessive birth weight of some of the infants and the hyperplasia of the islands of Langerhans in the fetal pancreas. This opinion is no longer tenable in the light of the evidence presented here. Fetal and neonatal deaths and the birth of unusually large babies occur with about the same incidence before diabetes becomes manifest as afterward. Hyperplasia of the islands of Langerhans, cardiac hypertrophy and excessive erythropoiesis in the fetal liver, which are found in the newborn of diabetic women, have also been demonstrated to occur in infants before symptoms or signs of diabetes could be recognized in their mothers. There is no reason for assuming that the factor or factors that cause fetal and neonatal deaths and that lead to the production of unusually large infants and the other anatomic changes in the viscera of the fetus during the years before diabetes can be diagnosed are not identical with those that are producing the same results after the syndrome has become established.

The birth of large babies and the frequent occurrence of fetal deaths before the appearance of diabetic symptoms has also been noted by Allen.[6] According to him "glycosuria is only another manifestation of a condition that may produce larger babies and sometimes kill them before birth." This is a vague statement and is far from being proved, since the nature of the "condition" remains obscure. There is no certainty even that the lethal factor for the fetus is identical either with the one that produces large fetuses or with the one that leads to the visceral changes in the fetus. In the present study no correlation could be found between the birth weight of the infant and the mortality. The number of deaths was no higher in babies that weighed over 4.5 Kg. than in those that weighed from 2.5 to 3.5 and from 3.5 to 4.5 Kg. Although the visceral changes described by Miller and Wilson apparently tend to occur more frequently in fetuses that weigh more than 4 Kg., the changes were also found in infants weighing considerably less. Furthermore, there were some women who, although they had several pregnancies before and after the onset of their diabetes, had none that resulted in the death of the fetus or newborn infant. Just why some fetuses escape and why the lethal factor does not repeat itself from one pregnancy to another require an explanation as much as the large number of fetal deaths. Two facts are clear from the present study. The lethal factor for the fetus may be in effect from fifteen to twenty years before the onset of diabetes can be recognized, and the effect of the lethal factor becomes greater as the mother approaches the onset of clinical signs and symptoms of the syndrome. Many other facts will be needed, however, before the relationship between fetal death, overweight fetuses and the eventual production of hyperglycemia in the mother is clarified. White and Hunt have stated that fetal death in the diabetic woman is associated with a high concentration of gonadotropic substance in the maternal serum during the latter part of pregnancy and that the administration of estrogens and progesterone can prevent the large number of fatalities.[7] This contention is not borne out by the investigations of the Smiths, who noted fetal and neonatal deaths in the presence of normal serum levels of gonadotropic substance in the diabetic mothers' bloods[8] and observed normal infants to be born in spite of decidedly elevated concentrations of gonadotropic substance in other mothers with diabetes.[9] Whether or not the administration of estrogens and progesterone to the mother during the latter months of pregnancy will prove effective in lowering fetal mortality must await further trial, since White's series of cases is too small to permit any definite conclusions. For obvious reasons the anterior pituitary has been considered by some as playing an important part in the production of large fetuses and the high incidence of fetal and neonatal deaths. Although the role of the anterior pituitary in the production of diabetes has recently received great emphasis, considerably more work must be done before the death of the human fetus and newborn infant and the excessive size

TABLE 5.—*Effect of Maternal Glycosuria Without Diabetes on Fetal and Neonatal Mortality.*

Case	Symptoms	Glycosuria	Highest Fasting Blood Sugar, Mg. per 100 Cc.	Dextrose Tolerance Curve	Infant	Birth Weight, Kg.	Comment
1	0	+	117	.....	Died 3d day	2.6	45,000 erythroblasts per cu. mm. of blood; large heart by x-ray; mother Rh+; later became diabetic
2a	0	+	116	Normal	Died 2d day	4.3	Atresia pulmonary valve
2b	0	0	100	.....	Stillborn	5.2	Cerebral hemorrhage at autopsy
3	0	+	93	Normal	Stillborn	5.0	Enlarged liver and spleen and erythropoiesis in liver at autopsy
4	0	+	Normal	.....	Lived	5.4	Large heart by x-ray; erythroblasts, 71 per hundred white blood cells
5	0	+	107	.....	Lived	3.7	
6	0	+	...	.....	Lived	2.7	
7	0	+	106	Normal	Lived	3.4	
8	0	+	...	.....	Lived	3.8	
9	0	+	101	Normal	Lived	4.2	
10	0	+	106	Normal	Lived	3.8	
11	Polydipsia polyuria	+	71	Normal	Lived	3.5	

6. Allen, E.: Glycosurias of Pregnancy, Am. J. Obst. & Gynec. **38**: 982, 1939.

7. White, Priscilla, and Hunt, Hazel: Prediction and Prevention of Pregnancy Accidents in Diabetes, J. A. M. A. **115**: 2039 (Dec. 14) 1940.

8. Smith, G. V., and Smith, O. W.: The Anterior Pituitary-Like Hormone in Late Pregnancy Toxemia, Am. J. Obst. & Gynec. **38**: 618, 1939.

9. Smith, O. W., and Smith, G. V.: Prolan and Estrin in the Serum and Urine of Diabetic and Nondiabetic Women During Pregnancy, Am. J. Obst. & Gynec. **33**: 365 (Oct.) 1937.

of the infant can be ascribed to this gland. It is true that the injection of extracts of the anterior pituitary lead to the production of postmature fetuses and a high fetal mortality in animals,[10] but there is no work as yet to suggest that the infants of diabetic mothers have an abnormally prolonged gestation period.

A large percentage of patients in the present study included women who could be classified as mildly diabetic. They had glycosuria, diabetic dextrose tolerance curves and high fasting blood sugars but did not require insulin. There has been a tendency in some clinics to minimize the importance of this group. The evidence presented here indicates that with respect to fetal mortality and the production of somatic and visceral changes in the fetus they are to be considered similar to the infants of women whose diabetes is severe and of long standing.

A special group of 11 mothers were observed who had glycosuria but who could not be diagnosed as diabetic, since the carbohydrate metabolism was otherwise within normal limits. The fact that the offspring of these women showed the same changes and the same poor chances for survival as did those born to mothers with outspoken diabetes indicates that glycosuria in the latter months of pregnancy is not to be regarded as "benign" for the fetus. The importance of even a single observation of dextrose in the urine is revealed in case 4 of table 5. During this particular pregnancy the mother had tests for sugar done on her urine on twenty different occasions, and reducing substances were found only once. The large number of tests were done because she had glycosuria for seven months prior to a previous pregnancy. The twenty tests reveal how completely the glycosuria had subsequently disappeared. In spite of the fact that the evidence of diabetes is practically nil during this pregnancy, the infant was found to weigh 5.4 Kg. at birth, had on roentgen examination an enlarged heart and was found to have 71 erythroblasts per hundred white blood cells in the peripheral blood—a clinical picture that is encountered with considerable frequency in babies of diabetic mothers. The fact that fetal death and pronounced somatic and visceral changes can occur in infants born to women with so little evidence of diabetes, and for many years before diabetes can be considered on any grounds, suggests that there may be occasions when fetal deaths occur and large babies are born and yet the mother may never develop the recognizable signs and symptoms of diabetes. The solution of the cause of fetal death and large babies in pregnant diabetic women may give us more light not only on diabetes but also on the causes of unexplained fetal death.

CONCLUSIONS

1. In pregnancies complicated by diabetes mellitus the fetal and neonatal mortality is about five times higher than that in nondiabetic pregnancies. The fetal and neonatal mortality is as high during the five years immediately preceding the onset of diabetic symptoms as after the syndrome has become established. An increased fetal and neonatal mortality can be observed from fifteen to twenty years before the clinical symptoms and signs of diabetes can be recognized.

2. Infants with a birth weight of 5 Kg. or more are born to women before they become diabetic with the same high frequency as after diabetic symptoms have appeared. The incidence of infants whose birth weight is 5 Kg. or more is about eighty times higher in pregnancies complicated by diabetes than in nondiabetic pregnancies.

3. The fetal and neonatal mortality is no higher among those infants who weigh 4.5 Kg. or more at birth than among those who weigh between 2.5 and 4.5 Kg.

4. In the presence of mild diabetes (not requiring insulin) and in the absence of maternal complications of pregnancy the fetal and neonatal mortality is four times higher than that in the nondiabetic population. Severe complications of pregnancy in women with diabetes increase the risk to the infant.

5. The presence of glycosuria in the last months of pregnancy in women whose carbohydrate metabolism is otherwise apparently normal is associated with a fetal and neonatal mortality that is as high as that among the offspring of women with definite diabetic signs and symptoms.

10. Cutuly, E.: Effects of Lactogenic and Gonadotropic Hormones on Hypophysectomized Pregnant Rats, Endocrinology **31**: 13, 1942. Teel, M.: The Effects of Injecting Anterior Hypophysial Fluid on the Course of Gestation in the Rat, Am. J. Physiol. **79**: 170, 1926. D'Amour, F. C., and D'Amour, M. C.: Studies in the Action of Estrin in Pregnancy, ibid. **105**: 26, 1933.

CLINICAL ASPECTS OF SHOCK

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The difficulties which beset the study of shock in wartime are obvious. Casualties, when they occur, are apt to be in overwhelming numbers, and the available clinical staff to deal with the cases is so overworked with urgent therapeutic procedures that adequate recording and study may be impossible. For this reason clinical data on shock are infinitely more scanty than reports on animal experimentation. In this school and hospital some 200 wounded patients were admitted during the London air raid period; of these, 28 had injuries of such severity as to require blood transfusion. In spite of considerable reduction of staff, circumstances were thus reasonably favorable for simple clinical observations and records to be made. It is on the basis of this experience that the opinions expressed in this paper are put forward.

When the present conflict began, views generally held of the nature of traumatic shock were those which had been reached at the end of the last war, with certain important modifications of outlook established by such experimental investigators as Blalock.[1] A fundamental feature of wound shock was taken to be a depleted circulating blood volume (oligemia). The investigators of the last war had made something of a mystery of the loss of blood, but since then the experimentalists had realized that probably the major part was lost by hemorrhage and exudation into the injured tissues.

CLASSIFICATION

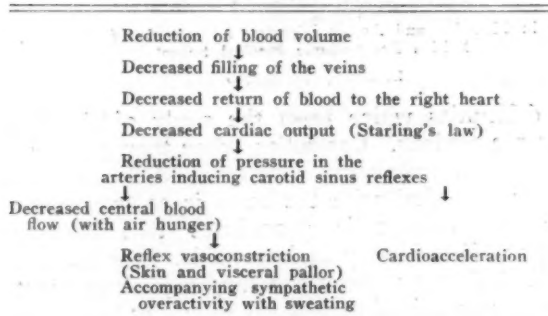
Any attempt to produce a complete clinical classification of shock can be only provisional, as it is not yet possible to include all the complex factors involved. Blalock's classification into hematogenic, cardiogenic,

The author's colleagues, particularly Drs. Bywaters and Sharpey-Schafer, have made continued efforts to work out these problems. Professor Grey-Turner and the surgical staff gave valued cooperation, without which the recorded observations could not be made.

1. Blalock, Alfred: Experimental Shock: Cause of Low Blood Pressure Produced by Muscle Injury, Arch. Surg. **20**: 959 (June) 1930; Surg., Gynec. & Obst. **58**: 551, 1934.

neurogenic and vasogenic has much to recommend it, but it may often break down in clinical practice owing to the lack of accurate knowledge of the complex interrelations of these factors in any given case. Elsewhere I [2] produced a clinical grouping of types of shock, including such varied causes as hemorrhage, vasovagal syncope, pulmonary embolism, bacterial toxemia, diabetic coma and the crises of Addison's disease. If one adds to this complex clinical series the various methods by which shock may be produced in animals, e. g. histamine poisoning, hypertonic salt injections, intraperitoneal muscle implantations, rapid hammering of limbs and battering in revolving drums, it is realized that the manifold types of insult which may produce circulatory collapse or shock are as numerous as the causes of pyrexia. Investigators in the last war made a fundamental error in accepting histamine shock as a basis for their clinical studies. To secure a balanced outlook on the war problem of shock, attention should be directed, not to the more esoteric laboratory technics, but to the phenomena of shock as it occurs in bomb, battle or civil injuries. Observations made on human clinical cases yield a sufficient number of leads for

Sequence in Oligemic Shock


Reduction of blood volume
Decreased filling of the veins
Decreased return of blood to the right heart
Decreased cardiac output (Starling's law)
Reduction of pressure in the arteries inducing carotid sinus reflexes
Decreased central blood flow (with air hunger)
Reflex vasoconstriction (Skin and visceral pallor) Accompanying sympathetic overactivity with sweating
Cardioacceleration

experimental work which, for purposes of war research, should be devoted to the extension and amplification of the detail which can be observed in man.

For practical purposes, at the present stage of our knowledge, the outstanding clinical parts of the problem of shock in war injuries are as follows:

1. Oligemia and its sequelae. This usually results from hemorrhage. In a few instances it may result from plasma loss into the traumatized tissues, but in my experience this is limited to burns and crushing injuries.

2. Renal failure following recirculation through tissues rendered necrotic by previous ischemia (e. g. crushing or compression injuries, traumatic arterial spasm).

3. Burns. These present a special problem of their own.

4. Complicating factors, e. g. infection and pulmonary fat embolism.

This article will deal particularly with the effects of oligemia resulting from hemorrhage, as this constitutes the major cause of 90 per cent of the cases of shock seen following war injuries.

The classic concept of oligemic shock may be represented by a sequence as presented in the accompanying tabulation. Hence the familiar picture of the patient with a pallid clammy skin, a rapid thready pulse with a profound fall in blood pressure in the more advanced stages.

2. McMichael, J.: Brit. M. J. **2**: 671, 1942.

This basic concept requires little modification, but the more detailed hypotheses of the physiologic mechanisms at work have required considerable amplification.

I. REDUCTION OF BLOOD VOLUME

The fundamental feature of oligemia was established in the last war by the pioneer work of N. M. Keith.[3] About the same time the experimentalists, who were using histamine to produce a shocklike condition in animals, became impressed with the possibility that this substance might play a major part in the production of shock. A hypothesis of traumatic toxemia was built up. Bayliss and Cannon [4] crushed the limbs of animals and found that a fall in blood pressure did not result so long as the blood flow through the affected limb was occluded. It was thought that histamine-like substances liberated from the damaged muscles escaped into the circulation and produced capillary paralysis, plasma loss and hemoconcentration. Clinically, evidence was produced for hemoconcentration in cases of injury by Cannon, Fraser and Hooper.[5] A survey of their data, however, shows some unexplained discrepancies. Most of the color indexes were surprisingly low. Thus in case 41 A the red cell count was 8 million with 80 per cent hemoglobin, giving a color index of 0.5. As we now know that low color indexes develop only some time after severe chronic hemorrhage, the only explanation is that some patients in this series had antecedent hypochromic anemia! None the less the traumatic toxemia theory held the field until the experimental work was further amplified, particularly by Blalock (1930) in America and by Holt and Macdonald [6] (1934) in this country. They demonstrated that the crushed limbs of animals swelled considerably as the result of local blood and plasma loss, which was quantitatively adequate to account for the fall in blood pressure. On looking further into the records from the last war, we find that the cases recorded clinically by Keith (30 well documented cases) practically all showed low hemoglobin values, most of them ranging from 75 to 85 per cent, when they first came under observation. Only 1 case of Keith's series (S. S., table IV) showed a hemoglobin of 125 per cent. This case, however, must obviously be excluded from the series of patients with wound shock, as there was no injury, and no satisfactory cause of death was found in the postmortem examination! The Medical Research Committee's reports also include other records indicating the importance of hemorrhage in producing the picture of shock. It is curious to read in the introduction to the Medical Research Committee's main publication something of an apology for including the reports of Bazett [7] and of Robertson and Bock [8] on hemorrhage.

3. Keith, N. M., in Reports of Special Investigation Committee on Surgical Shock and Allied Conditions: IX. Blood Volume Changes in Wound Shock and Primary Hemorrhage, Medical Research Committee, Special Report Series, No. 27, London, His Majesty's Stationery Office, 1919.

4. Bayliss, W. M., and Cannon, W. B., in Reports of the Special Investigation Committee on Surgical Shock and Allied Conditions: VIII. Traumatic Toxemia as a Factor in Shock, Medical Research Committee, Special Report Series, No. 26, London, His Majesty's Stationery Office, 1919.

5. Cannon, W. B.; Fraser, J., and Hooper, A. N., in Reports of Special Investigation Committee on Surgical Shock and Allied Conditions: Wound Shock and Hemorrhage, Medical Research Committee, Special Report Series, No. 25, London, His Majesty's Stationery Office, 1919.

6. Holt, R. L., and Macdonald, A. D.: Brit. M. J. **1**: 1070, 1934.

7. Bazett, H. C., in Reports of the Special Investigation Committee on Surgical Shock and Allied Conditions: Wound Shock and Hemorrhage, Medical Research Committee, Special Report Series, No. 25, London, His Majesty's Stationery Office, 1919.

8. Robertson, O. H., and Bock, A. V., in Reports of Special Investigation Committee on Surgical Shock and Allied Conditions: Wound Shock and Hemorrhage Medical Research Committee, Special Report Series, No. 25, London, His Majesty's Stationery Office, 1919.

Hemorrhage is held to be different from "shock from more gradual depletion of the circulation and impoverishment of the blood." Keith was made personally responsible for regarding "the two factors of shock and hemorrhage . . . as practically equivalent."

From the historical point of view, therefore, we may note and learn that good clinical evidence of the last war was forced into the background in favor of speculation based on animal experimental work. Hemoconcentration came to be regarded as a fundamental feature to be aimed at in the production of experimental shock, and in the laboratory shock was produced by a variety of measures which in no way resembled the injuries of war or civil accident; e. g. the intraperitoneal insertion of muscle and the intramuscular injection of hypertonic salt solution. Such experiments, while providing much information of academic interest, appear to be well off the main line of approach to the problem of shock in war injuries.

The Clinical Evidence of Hemorrhage.—The survey made by Grant and Reeve [9] of collected material from the London air raids indicates that the hemoglobin value in patients with a low blood pressure is frequently reduced and is often about 80 per cent or below. They found no evidence of hemoconcentration except in 1 case probably to be regarded as a crushing injury. In some cases, however, the hemoglobin figure could be regarded as normal. It should be noted that Blalock [1] has found that blood dilution may fail to occur in hemorrhage. Kekwick, Marriott, Maycock and Whitby [10] in a series of air raid casualties also found no evidence of hemoconcentration, although some of their cases showed normal hemoglobin figures.

It should be emphasized that most of the air raid casualties were observed in hospitals within an hour of receiving their injuries. For this reason it is important to consider the rate at which hemodilution may occur in normal persons who have been subjected to blood loss. The results of animal work in this respect have been misleading. There is an impression that proportionate hemodilution occurs almost *pari passu* with bleeding. While this may occur in experimental animals, studies on the rate of hemodilution in human volunteers made by Wallace and Sharpey-Schafer [11] showed that, after a loss of 20 to 25 per cent of the blood volume, full hemodilution was not achieved until thirty-two hours, on the average, had elapsed. The initial dilution within one hour was seldom more than 10 per cent. Thus in wounded patients who have hemoglobin values of 100 per cent or over it is always possible that their initial hemoglobin value may have been 10 per cent higher. This appreciation of slow dilution following hemorrhage in man is no new discovery. In the last war Robertson and Bock quoted Govaerts, who recorded that "the red cell count decreased progressively until about the third day following primary blood loss." Robertson and Bock emphasized that the hemoglobin reading immediately after an injury may thus be a poor index of the severity of the bleeding. It is only the later dilution which gives some indication of the degree of blood loss. The conclusion, therefore, is that hemoglobin values around 90 per cent are the rule within the first hour of sustaining injuries, no matter how severe the associated hemorrhage has been.

9. Grant, R. T., and Reeve, E. B.: Brit. M. J. **2**: 293 and 329, 1941.
10. Kekwick, Alan; Marriott, H. L.; Maycock, W. d'A., and Whitby, L. E. H.: Lancet **1**: 99, 1941.
11. Wallace, J., and Sharpey-Schafer, E. P.: Lancet **2**: 393, 1941.

Further evidence pointing to hemorrhage as the most important factor in the usual case of "shock" include: 1. The history. Sometimes when external blood loss from a major injury appears to be slight, interrogation of the patient not infrequently reveals some source of hemorrhage from a relatively slight injury which might otherwise be overlooked. For example, a patient admitted with severe injuries of the limbs (compound fractures) volunteers the information that in the ambulance his pillow had been soaked with blood from a scalp wound. 2. Physical examination. Gross swelling of the soft tissues in a wounded part can accommodate considerable quantities of blood. The volume of the thigh regarded as a cylinder 30 cm. in length and 14 cm. in diameter is 4,624 cc. If the diameter of the same thigh is increased by blood effusion to 16 cm. (volume 6,027 cc.) the increase in volume is 1,403 cc. In the presence of swelling due to multiple wounds it can readily be appreciated that a large proportion of the blood volume may be lost into the tissues. 3. The clinical course. The response to early and adequate

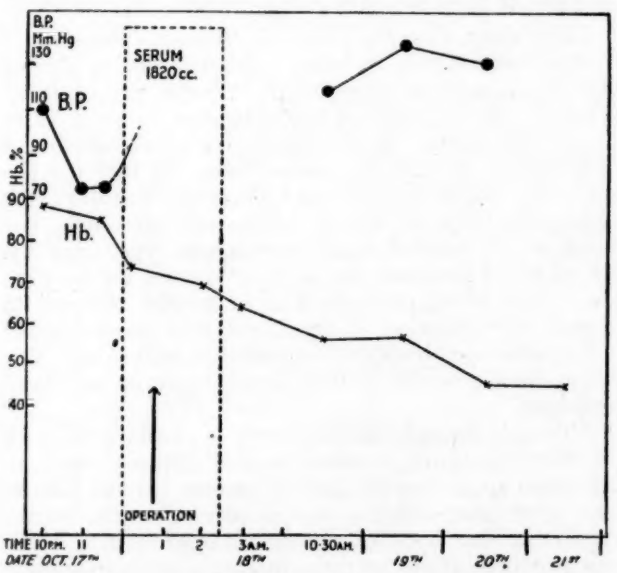


Chart 1.—Record in case 1. Note initial fall in blood pressure while the patient was heated, the satisfactory response to massive serum transfusion and the slow dilution of hemoglobin to a final level of 47 per cent. (By permission of the Edinburgh Medical Journal.)

transfusion makes it evident that patients in very profound shock may recover completely when the blood volume is restored, provided the wounds can be adequately dealt with and further hemorrhage arrested. The 2 following case records show the type of observation from which such conclusions may be drawn:

CASE 1 (chart 1).—A man aged 32 was admitted soon after injury with a compound fracture of the right ankle, swelling of the left thigh with a punctured wound, and a punctured wound of the arm. External hemorrhage was negligible. His condition on admission was quite good, the blood pressure being 110, the pulse rate 100 and hemoglobin 86 per cent. He was treated with warmth, morphine and raising the foot of the bed, but after one hour the blood pressure fell to 70, the hemoglobin remaining much the same. Owing to the arm wound the blood pressure could not be followed all the time, but by palpation there was clearly a rise of blood pressure after a liter of serum. The level of the hemoglobin after serum suggested that hemorrhage had probably occurred; and so it proved, as incision into the thigh revealed a large quantity of blood in the thigh and buttock. The further hemodilution gives some idea of the extent of the original hemorrhage, probably about half the blood volume.

CASE 2 (by permission of the director of medical service, Royal Canadian Army Medical Corps).—A soldier during exercises was accidentally thrown from his motorcycle under a tank. Treatment in the hospital was begun within one hour. His injuries consisted of a compound fracture of the humerus, fractures of the ninth, tenth and eleventh ribs and also of the second, third and fourth lumbar transverse processes, a ruptured right kidney, a simple fracture of the right ankle, together with concussion and scalp lacerations. His blood pressure remained unrecordable until 2 liters of plasma and blood had been given; 3.5 liters brought his blood pressure to 100 mm. of mercury at the end of two hours. At the end of four hours, when 5.5 liters of blood and plasma had been given, he was fit for operation. His wounds were excised, the fractures were reduced and the abdomen was opened to make sure of the absence of intraperitoneal hemorrhage. A large retroperitoneal hematoma was present, which was left alone. During the operation a further liter of blood was given. The patient made an excellent recovery and was up in the ward in ten days.

It is unnecessary to seek any cause for the collapse in these cases other than the hemorrhage, which is quantitatively adequate to account for the clinical state, quantitative replacement in each case being followed by a satisfactory outcome.

Circulatory Collapse Due to Plasma Loss.—As I have already stated, circulatory collapse due to plasma loss is a rare occurrence except in burns and crushing injuries. As a result of tissue trauma in these cases the "permeability" of the capillaries is altered and a fluid closely resembling plasma leaks out into the tissues. In some of our cases of crushing injury the hemoglobin rose as high as 140 to 160 per cent. The shock which resulted is not of a special type, since the fall in blood pressure was fully accounted for by fluid loss. The blood pressure was invariably restored to normal by redilution of the blood to a more normal hemoglobin level by plasma or serum transfusions. The fall in blood pressure in these cases, however, was very profound.

Although the circulatory effects of crushing injuries are relatively simple in their essential features, the more important sequela of this type of trauma is renal failure. This syndrome, which is now usually called the "crush syndrome," has been fully described elsewhere.[12] Various products from necrotic muscle (myohemoglobin, potassium phosphate and other substances) leak out into the circulation when this is reestablished locally following release from the compressing injury, and severe renal impairment with death in uremia follows in 66 per cent of the cases.

II. HEMODYNAMIC SEQUELAE OF OLIGEMIA

The sequence of events postulated by the physiologic principles that have been outlined has been generally substantiated. There are, however, some important modifications requiring emphasis:

(*a*) *Hypertensive Reactions.*—In a small proportion of wounded subjects, probably about 5 per cent, the blood pressure may be found to be raised on first examination. This reaction was noted by Grant and Reeves in their survey, and we also observed it in this hospital in 3 of our 28 cases of shock. The injuries sustained by these patients have often been severe: they look pallid and the radial pulse at the wrist may be of small volume. When the blood pressure is taken, however, the observer is surprised to find that it is raised to 150 or 160 mm. of mercury. Such observations are of importance, as it is known that these patients may subsequently pass into the more typical picture of oligemic collapse. Thus they must be very carefully watched in the hours succeeding admission. We have no satisfactory observations which throw light on the nature of this reaction, and it is mere speculation to suggest that the blood flow through the kidneys might be reduced and the hypertensive reaction results from the liberation of renin into the circulation.

(*b*) *Hypotensive Reactions.*—Generally speaking, blood losses up to a liter are well tolerated by normal persons, as far as can be gaged by blood pressure records. As long as such patients remain recumbent the signs of shock are not clinically obvious, apart from slight acceleration of the pulse (e. g. 60 to 80) and reduction of blood pressure by about 10 mm. of mercury. By the method of Cournand and Ranges[13] Dr. Sharpey-Shafer and I made further observations on a human volunteer on cardiac output and right auricular pressure in hemorrhage. During hemorrhage (900 cc.) when the blood pressure remained within normal limits, though falling slightly, the pressure in the right auricle was falling steadily (by 6 cm. saline). Along with this there was a parallel fall in the output of the heart to two-thirds the normal level (from 4.85 to 3.2 liters per minute). The blood pressure is thus maintained only by a considerably increased peripheral resistance. During this stage of hemorrhage it is to be noted not only that there is a considerable reduction of the volume of blood in the systemic venous system (low venous pressure) but that there is also evidence of a reduction of the volume of the pulmonary vascular bed.[14] Further observations of this type are still required in profound posthemorrhagic collapse.

Vasovagal Collapse (?carotid sinus syncope).—It was frequently noted in the blood transfusion depots that blood donors might faint during the withdrawal of the usual 380 to 540 cc. of blood.[15] At first it was thought that this type of faint might be emotional, due to fear, the sight of blood or the like. Wallace and Sharpey-Schafer, however, showed that, when collapse occurred owing to the loss of 1,000 to 1,200 cc. of blood, it was usually of this vasovagal type in which the fall of blood pressure is accompanied by a pronounced slowing of the heart rate. In the course of the observation mentioned in the preceding paragraph a vasovagal faint occurred after blood loss of 900 cc. The blood pressure fell from 110 to 58 and the pulse from 82 to 50. During the faint the right auricular pressure rose by 2 cm. from the low level reached at the end of the hemorrhage. The cardiac output also rose slightly (3.2 to 3.6 liters per minute). Subsequent intramuscular injection of 30 mg. of N. methyl amphetamine[16] restored the blood pressure from 60 to 135 mm. of mercury in eight minutes with little significant change in right auricular pressure or pulse rate, but the cardiac output rose from 3.6 to 4.8 liters per minute. Analysis of these data shows that vasovagal collapse is due to a sudden loss of peripheral resistance (presumably arteriolar tone) and, since there is an accompanying slowing of the pulse, there may be reason for suggesting that it is due to a hyperactive carotid sinus reflex. This speculation suggests that the threshold of sensitivity of the whole carotid sinus reflex mechanism is altered so that the sinus becomes sensitive to normal pressure, responding to it as though the pressure were raised. Professor

12. Bywaters, E. G. L.: Brit. M. J. **2**: 643, 1942.

13. Cournand, A., and Ranges, H. A.: Proc. Soc. Exper. Biol. & Med. **46**: 462, 1941.
14. Glaser, E. M., and McMichael, J.: Lancet **2**: 230, 1940.
15. Greenbury, C. L.: Brit. M. J. **1**: 253, 1942.
16. Supplied to us for testing by Burroughs Wellcome & Co. under the trade name of "Methedrine."

R. J. S. McDowall[17] has demonstrated an increased sensitivity of the carotid sinus reflexes after hemorrhage in animals. He believes that the sensitivity of the carotid sinus mechanism is disturbed by reduction of pressure in the great veins near the heart.

It is important to recognize that vasovagal collapse with a slow pulse can occur as a sequela of considerable hemorrhage as well as of small blood loss. The condition may recover spontaneously in the recumbent position and, as I have indicated, it is in this type of collapse that N. methyl amphetamine may prove of greatest value by its vasoconstrictor action.

The Relation of Blood Volume to Blood Pressure.—Apart from the occurrence of a vasovagal type of collapse, acute blood loss up to a liter may be well tolerated so far as blood pressure readings are concerned. With larger losses data regarding blood volume change are more scanty and the methods less certain. Kekwick and his collaborators considered that the blood volume might often be reduced as low as 2 liters in cases of severe shock. We have estimated blood volume in various ways[18] and we have also estimated blood loss in certain cases of gross hemorrhage, e. g. by estimating blood lost in the peritoneal cavity in a case of traumatic rupture of the liver. We believe that when shock is profound (systolic blood pressure lowered to 60 mm. of mercury or less) the circulating blood volume has often been reduced to 2 liters or below. This opinion is substantiated by the quantities of blood or plasma required to bring the blood pressure back to normal. This relationship of blood volume and blood pressure in hemorrhagic shock differs considerably from the concept put forward by Keith that shock was profound when the blood volume was reduced to 60 per cent of normal. This is of importance in giving us a clear idea of the volumes of blood, serum or plasma which have to be transfused in order to restore the blood pressure in wounded patients.

Recent work by McMichael, Sharpey-Schafer, Mollison and Vaughan[19] seems to show that chronic reductions of blood volume (severe anemias) to the level of 2 liters are not uncommon without vascular hypotension. If our views are correct, the possibility of searching for a pharmacologic remedy for shock must again be considered possible. It is probable that such a drug will prove to be a venoconstrictor substance, reducing the peripheral capacity of the small veins and thereby maintaining the pressure near the right side of the heart.

Pulse Rate.—The conception of an increasing pulse rate running inversely to the fall in blood pressure and cardiac output has required modification. The conflicting mechanisms in the blood pressure response to hemorrhage already emphasized may possibly account for certain anomalies of pulse behavior. Sometimes the pulse is rapid but it is seldom above 120 per minute, while at other times it is slow in the presence of a low blood pressure, e. g. 80 per minute. Chart 2 gives a record of case 3, indicating the anomalous reactions which may be encountered in treatment. A boy aged 12 years had one kidney ruptured by a lorry wheel three hours before infusion was begun. Collapse was profound and the pulse at first unrecordable. The improvement due to the first transfusions of blood and serum was not maintained, and shock again increased parallel with concentration of the blood. Two doses of four times concentrated serum caused improvement, which was maintained. There was dilution of the blood, and the blood pressure rose. Note the increasing pulse rate during and after recovery of blood pressure.

The moral is that pulse rate is far less reliable than blood pressure in serving as a guide for estimating either the severity of a given case of shock or the results of treatment.

III. TREATMENT

The classic lines of therapy include rest in the recumbent position, warmth and morphine. With the first there can be no argument.

The application of warmth, however, has come in for some criticism. It is generally known that the warm skin may accommodate up to half a liter of blood, and when the vital organs are suffering from a depleted circulation it seems unwise to deflect this valuable fluid into the temperature regulating vessels of the surface. Considerable support for the idea that heat may be harmful comes from the work of Blalock and Mason,[20] who showed a very significant decrease in the survival

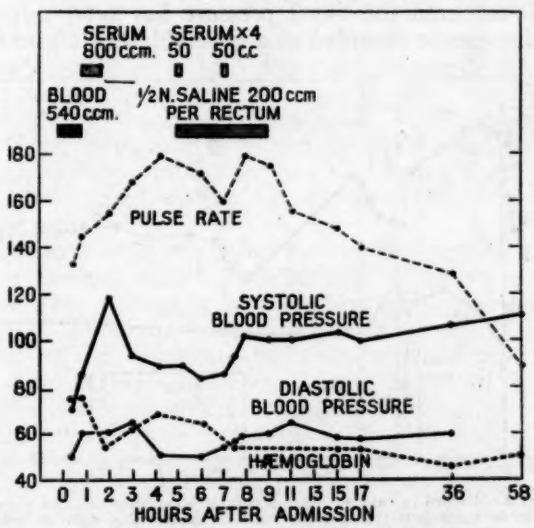


Chart 2.—Record in case 3. Note the increasing rapidity of the pulse with recovery. Total volume of transfusion equal to 60 per cent of blood volume in a boy weighing 60 pounds (27 Kg.). (By permission of the Lancet.)

period of dogs which were heated after the induction of hemorrhagic shock. It seems possible from some of our records that overheating may have been harmful. The patient illustrated in chart 1 was heated between 10 and 11 p. m., and it was during this time that he developed a fall in blood pressure. It is felt, however, that much more work is needed on the influence of cold and heat before heating appliances should be completely forbidden. We take it as accepted, however, that excessive heating is now regarded as harmful and that we should limit our measures to the application of enough warmth for comfort until we know more.

Morphine is in no sense a remedy for shock. Shocked patients do not usually complain of pain, and there is little need to give them morphine unless they do. We frequently observed that the injured patients became restless and began to complain of the pain of their wounds when the blood pressure was recovering. Morphine could then be given, if necessary intravenously, by injecting into the infusion tubing, in the usual dosage.

17. McDowall: Personal communication to the author.
18. Bushby, S. R. M.; Kekwick, Alan, and Whitby, L. E. H.: Lancet **2**: 540 (Nov. 2) 1940. Hill, D. K., ibid. **1**: 177 (Feb. 8), 335 (March 15) 1941.
19. McMichael, J.; Sharpey-Schafer, E. P.; Mollison, P. L., and Vaughan, J.: Lancet **1**: 637, 1943.
20. Blalock, Alfred, and Mason, M. F.: Comparison of Effects of Heat and Those of Cold in Prevention and Treatment of Shock, Arch. Surg. **42**: 1054 (June) 1941.

Transfusion, however, is the sheet anchor of treatment. Blood is the fluid of choice, but serum or plasma may be used equally well so long as excessive hemodilution is not produced. It was our practice to begin all urgent cases with serum, as this saved time in typing and cross matching which could be done later. There is no need to discuss the indications for transfusion in the cases of severe collapse. If the blood pressure is decidedly reduced, transfusion should be begun without hesitation. A critical level, however, is difficult to define; we have had experience of cases with blood pressures reduced to 80 to 90 mm. of mercury by hemorrhage. When left at this level for more than an hour or two, an irreversible and fatal collapse developed. It should be noted that there are other types of collapse in which blood pressures below 80 are well tolerated for long periods, e. g. coronary thrombosis. None the less I believe that, if the blood pressure does not show signs of recovering spontaneously from pressures below 90 at the end of one hour, transfusion should be begun without further delay.

The volume of fluid to be given can be estimated only by the blood pressure response. Transfusion must be continued until the blood pressure has been restored to what can be regarded as a normal level. Up to this

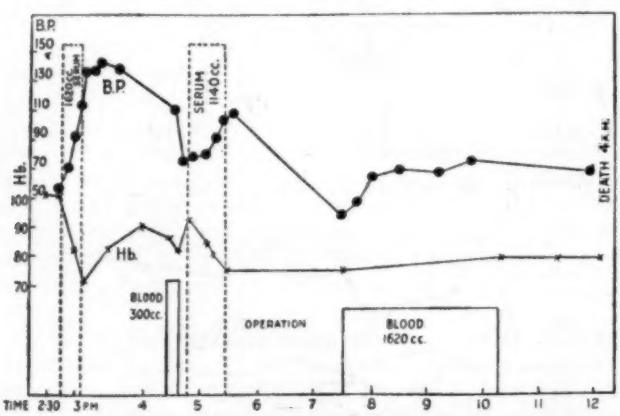


Chart 3.—Record in case 4. Note progressive decrease in blood pressure response to equivalent transfusions until an irreversible state is reached with no recovery. Optimum time for operation should have been after the first transfusion. (by permission of the Edinburgh Medical Journal.)

point it may be given as quickly as fluid will run into the veins; thereafter it should be slowed to a drip, the drip being maintained until the end of any necessary operation.

Other Measures in Treatment.—Oxygen therapy by the efficient B. L. B. mask was considered but had to be held in reserve. In our experience it was not found practical to do all that had to be done for a severely injured patient (make frequent blood pressure recordings, carry out the necessary examination, take the history, maintain a blood transfusion and so on) and give oxygen at the same time. Recent work suggests that oxygen therapy has only a limited value.[21]

Drug Treatment.—Vasoconstrictor drugs have so far not been considered to have any place in the treatment of shock. The administration of such substances was thought to be contraindicated, as vasoconstriction is a prominent feature of most cases of oligemic collapse. The most recent reports on N. methyl amphetamine [22] indicate that, while it may be of value in postoperative states, it is none the less essentially an arteriolar constrictor and as such has little place in the treatment of oligemic collapse. Where oligemia is complicated by a loss of arteriolar tone, as in the vasovagal cases or after anesthesia, it has already been shown to be of considerable value. I feel that there is an undoubted place for a substance which will diminish the capacity of the small venules in the periphery, as this mechanism is probably responsible for maintaining blood pressure in cases of severe anemia in which the blood volume is reduced.

21. Frank, H. A., and Fine, J.: J. Clin. Investigation **22**: 305, 1943.
22. Dodd, H., and Prescott, F.: Brit. M. J. **1**: 345, 1943.

IV. THERAPEUTIC FAILURES AND ANOMALOUS CASES

The early and adequate application of the foregoing therapeutic measures will be successful in the majority of wounded subjects. In a certain number, however, the treatment may fail, and the causes of this failure require analysis. In our own material in this hospital during the London air raids therapeutic failures were mostly accounted for when full postmortem analysis was made. Among the factors responsible are the following:

1. Continued hemorrhage and visceral injuries. In order to avoid this complicating factor an early and adequate review and assessment of the patient's injuries is necessary. In 1 of our cases a ruptured spleen had not been diagnosed clinically, while in another multiple rib fractures were associated with pleural and pericardial hemorrhage with cardiac tamponade.

2. Pulmonary fat embolism. This is another factor which may cause or increase the degree of collapse.

3. Superadded bacterial infection. This may result in (?) vasomotor paralysis from bacterial toxemia. This may play a part in the production of collapse in abdominal injuries, particularly those associated with perforation of the large intestine.

4. Preexisting disease. In raids on the civil population all ages are likely to be involved, and old age, for example, may change the picture considerably. In 1 middle aged patient a coronary thrombosis developed during a period of low blood pressure due to oligemic shock. Cardiac failure developed during the attempts which were made to increase the blood pressure by transfusion, but the patient ultimately made a good recovery.

5. Carbon monoxide poisoning. It should always be kept in mind that explosions in big cities may lead to the rupture of gas mains and that explosive gases by themselves in confined spaces may produce carbon monoxide poisoning. Several cases of this type were admitted to this hospital during the London air raid period. The blood pressure may be low on admission, and the case may be mistaken for one of wound shock.[23] Sir Cuthbert Wallace [24] reported a case which was probably of this type in the last war and which has been frequently quoted to emphasize the mysterious nature of shock. "One [person] was buried by the explosion of a shell in a cellar. He exhibited all the classic symptoms of shock, which lasted over forty-eight hours. . . . Treatment was of no avail." The postmortem examination did not show any gross lesion. We feel in retrospect that this case was probably one of carbon monoxide poisoning.

6. The irreversible state. It is now well recognized that if the blood pressure is reduced too low for too long as the result of hemorrhage, replacement of all the

23. McMichael, J., and Ruskin, H. D.: Lancet **2**: 677, 1941.
24. Wallace, Cuthbert: M. R. C. Reports of Special Investigation Committee on Surgical Shock and Allied Conditions: VIII. Traumatic Toxemia as a Factor in Shock, Medical Research Committee, Special Report Series, No. 26, London, His Majesty's Stationery Office, 1919.

blood which has been lost will not be followed by recovery. Chart 3 illustrates the development of the irreversible state in a patient who was treated for profound collapse following a bomb injury.

CASE 4.—A woman aged 56 was admitted after severe crushing injuries involving a compound fracture of the left leg; 1,600 cc. of serum was given, and the blood pressure rose to 140/90, with a fall of hemoglobin from 102 per cent to 72 per cent. An hour and a half later the hemoglobin had risen to 90 per cent again, which meant that the serum given had been almost completely lost from the circulation and the blood pressure had collapsed to 75 mm. of mercury. After further transfusion the patient was operated on, the wounds being excised and the leg put up in plaster. It was found that the skin of the leg had been torn from the underlying fascia and a large pool of blood had formed, containing several pints. In spite of further postoperative transfusion this patient died.

It is to be noted that the response to transfusion of equivalent quantities of blood on three successive occasions in this patient became progressively less. The final irreversible state which was reached after the operation was accompanied by considerable spasm of the veins, which made transfusion of the blood slow and difficult. This venospasm is not infrequently encountered in severe collapse, and it may be impossible to force blood into the veins even under pressure.

The nature of this irreversible state is not yet understood. It would be valuable if experimental investigators could concentrate on an analysis of this phenomenon. Wiggers and Werle [25] believe that it is due to a central cardiac failure resulting from prolonged coronary ischemia. Further electrocardiographic studies might be helpful in clearing up this point. It seems that under battle conditions this irreversible state may constitute the major problem of shock owing to the unavoidable delays in getting the patients back to base hospitals. The problem is therefore both urgent and practical.

V. THE ORGANIZATION OF RESUSCITATION TEAMS

It is obvious from the foregoing that the management of the severely injured patient requires as continuous and assiduous attention as the care of diabetic coma. Under the conditions in which these casualties occur, the technical skill of the surgeon and his assistants had to be fully utilized in the operating theater. It is therefore important that physicians trained in transfusion technics should cooperate with the surgeons in charge of special resuscitation wards. The patients who are admitted in batches are reviewed quickly by the surgeon and physician together: throughout their care the surgeon remains in charge, but it was our practice to leave the decisions regarding the urgency of operation and modifications of the order of priority in the operating theater to the physician. He had the assistance of the surgeon between operations when decisions were difficult. Repeated blood pressure observations are necessary in the control of these patients, and in civil hospitals the help of students was of great value. Under military conditions it may be that orderlies and nurses should be trained in the recording of blood pressures. If possible one nurse or orderly should be allocated to each patient, or at most 2 patients, requiring transfusion. This means a considerable expansion of the ordinary estimate of nursing requirements. It was only when these points were realized that we achieved greater success in our treatment of shocked patients.

Throughout the foregoing description I have emphasized the part which may be played by the physician in charge of resuscitation work. It must never be forgotten, however, that the patients have injuries requiring surgical attention, and shock cannot be treated without careful dovetailing of the medical measures of resuscitation to the necessary surgical interventions. Even patients with their blood pressures restored to normal may deteriorate by failure to operate at the opportune moment. Generally speaking, the longer operation is delayed, the worse for the patient, and when shocked patients happen to be admitted singly there is a good deal to be said for carrying out resuscitation measures in the operating theater just prior to and during operation. Delay in operation leads to such complications as infection, further hemorrhage and the development of the irreversible state.

Ducane Road, London, W. 12.

25. Wiggers, C. J., and Werle, J. M.: Am. J. Physiol. **136**: 421, 1942.

PES PLANUS

MILITARY SIGNIFICANCE AND TREATMENT WITH SIMPLE ARCH SUPPORT

MAJOR FREDERIC W. ILFELD
MEDICAL CORPS, ARMY OF THE UNITED STATES

Prior to the declaration of war, the degree of pes planus was of prime importance in determining the pedal physical qualifications for military service. Regulations of both the Army and the Navy were definite regarding the type of foot acceptable, and persons with pronounced or third degree pronation were eligible for discharge or disqualification without consideration of the clinical symptoms.[1]

In many instances deserving men were denied admission into the service, and, perhaps in too many cases, men were discharged because their feet appeared to be "flat." Approximately 50 per cent of the patients examined by the Certificate of Disability Discharge board at the Station Hospital, Camp Callan, California, appeared for orthopedic complaints, and of these one third were for foot complaints. Those discharged from the Army for foot symptoms constituted 40 per cent of the total receiving a medical discharge for the period from March 8 to June 1, 1941 and 28 per cent of the total from June 1 to Oct. 1, 1941.

Since December 7, 1941 a firmer attitude in regard to pes planus has been assumed by the Selective Service and Certificate of Disability Discharge examining boards. Only 2.6 per cent (13 cases) were discharged by the Certificate of Disability Discharge board for pes planus during the year 1942, and 7.3 per cent (67 cases) were reclassified for limited service. But foot complaints continue to be a major military problem, as shown by the report of Berkman,[2] who states that 40 per cent of the complaints in the orthopedic clinic at Camp Lee, Virginia, are referable to the foot.

Studies undertaken at the Station Hospital, Camp Callan, California, during the fall of 1941 justify this change in policy. Complete histories of the men and examinations of their feet (table 1) were recorded on 75 white soldiers who were admitted to the orthopedic service with foot complaints and subsequently discharged from the service in accordance with existing army regulations. These results were compared with the findings obtained on examination of 347 white soldiers with no subjective symptoms. In addition, foot prints

1. Medical Department: Standard of Physcial Examination for Entrance into the Regular Army, National Guard and Organized Reserves, Army Regulations 40-105, United States War Department, section XIX, paragraph ab; also MR 1-9, C1, paragraph 46 j.

2. Berkman, E. F.: Etiological Possibilities of March Fractures, J. Bone & Joint Surg. **25**: 206 (Jan.) 1943.

were made by blackening the soles with finger print ink and then obtaining an impression with full weight bearing (fig. 1). As is customary, the weight bearing line as tested by a plumb line was noted. A perpendicular to the line connecting the first metatarsal head and os calcis gave the measure of pronation.

Table 2 shows that the majority of the men receiving discharges had a so-called third degree pes planus. It is significant to note that 7 (2 per cent) of the normal group also had third degree pronation and that 52 (15 per cent) had moderate or second degree pronation. It follows, therefore, that pronounced pronation may be present without symptoms. In fact, one of the most severe cases of pes planus noted in this series was that of a soldier who had been able to march 20 or more miles daily with full pack and no complaints. These findings substantiate Lewin's [3] statement that "the height of your arch does not determine the strength or usefulness of your foot." Quite often cases of pes cavus, that is, those with pronounced elevation of the longitudinal arch, give considerable pain, especially over the metatarsal heads. "A flat foot may give a good performance, whereas a high-arched foot may be incompetent." [3]

TABLE 1.—*Pes Planus Survey*

Name: .....
Age: .....
Serial No.: .....
Station: .....
Home Address: .....
Occupation: .....
Steady ..... Intermittent .....
Site of pain: .....
Sharp..... Dull..... Constant..... Intermittent.....
Duration.....
Worn arch supports before.....
Backache.....
Fatigue..... Pain in knees..... Metatarsalgia.....
Other symptoms: .....
Length of Army service:..... No. of hikes..... Length.....
Ability to drill:.....
Other symptoms:
Examination: Degree of pronation..... Right..... Left.....
Rotation of os calcis.....
Callosities.....
Corns.....
Hammer toes.....
Weight..... Height.....
Diagnosis: .....
Treatment: .....
Disposition: .....

The symptoms of foot strain from pes planus are subjective. However, several factors in the patient's history are helpful in evaluating these symptoms. For example, of the 75 discharged, 48 (64 per cent) gave a history of previously using supports; an association of backache was noted in 46 cases (61 per cent) and pain in the knees in 54 (72 per cent). Only 1 patient was overweight, although the weight and height were carefully checked in each instance. No significant difference was noted in the relationship of former occupation, whether sedentary or active (table 3). The age limits in the two groups were comparable.

Efforts to relieve symptoms with "Thomas heel," a combination of heel and sole elevations, or "Jones metatarsal bars," were usually unsatisfactory. Long and frequent marches soon render these elevations of the shoe ineffective, whereas an arch support in the shoe is more durable. Moreover, arch supports can be changed from shoe to shoe and are easily raised or lowered. After various experiments I worked out a simple and practical arch support. It is effective in relieving symptoms in the majority of cases, easy to make, inexpensive and adjustable.

3. Lewin, Philip: The Foot and Ankle, Philadelphia, Lea & Febiger, 1941.

To make the support cut a piece of leather (fig. 2) about 7½ inches long, 4 inches wide at the instep, tapering down to about 2 inches at the heel and about 4 inches in the metatarsal region, depending on the size of the soldier's foot. The leather beveled on the lateral edge gives the effect of a Thomas heel. Adjacent to

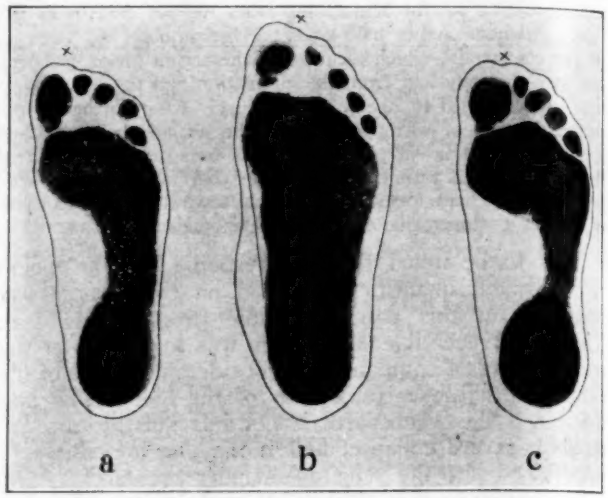


Fig. 1.—*a*, normal foot print; *X* marks the weight bearing line. *b*, third degree pes planus; note line connecting matatarsal head and os calcis, enabling measurement of degree of pronation. This type of foot may or may not be symptomatic. *c*, moderate pes cavus, more likely to cause symptoms than *b*.

the longitudinal arch the leather is skived to allow for molding. The front of the support may be beveled for comfort. Soak the leather for several hours in water and then fasten onto a stock plaster with gauze bandage and allow to dry overnight into the shape of the plaster model. The plaster cast can be cut, deepening the longitudinal and metatarsal arches as indicated. Shape a pad of orthopedic felt, ordinarily used for padding plaster casts, to give support under the longitudinal arch and, if needed, under the metatarsal arch. Fasten this pad with glue to the leather. The support can be raised

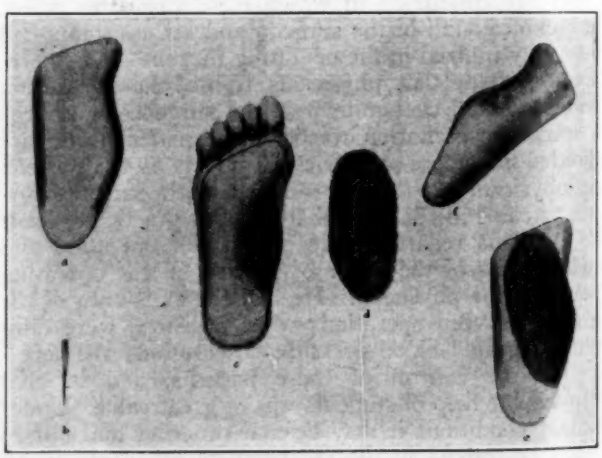


Fig. 2.—*a* and *b* indicate shape of leather with skiving on the lateral side giving the effect of a Thomas heel. *c*, wet leather bandaged onto plaster model and left to dry overnight. *d*, felt pad is shaped by emery wheel or razor blade to afford support under longitudinal and metatarsal arches. *e* and *f* illustrate completed arch support.

or lowered by the addition to, or cutting away of, the felt pad. If the felt pad packs down to where the support is too low, it may be raised by adding more felt.

Several different sizes of molds are made—large, medium and small—in order that supports may be fitted

to the patient on his first visit to the clinic. Approximately 2 to 3 hundred pairs per month have been prescribed in the orthopedic clinic at Camp Callen, California. These arch supports cost about 20 cents a pair and are light and flexible.

Of course no orthopedic aid can be effective without a proper fitting shoe. Length of the sole should allow a thumb's breadth from the great toe to the end of the shoe with full weight bearing, preferably tested with the soldier carrying a pack (50 to 70 pounds). In many instances shoes provided have been too short. For determining the width of the shoe the forefinger should be able to be inserted between the great toe and the tongue of the shoe [3] or by determining the amount of stretch of the leather at the level of the metatarsal heads. A shoe in the latter test that is too wide will allow a crease and if too narrow will present no elasticity.

Common aids in foot hygiene include the lacing of shoes loosely at the bottom, snug in the middle and loose at the top. This affords support to the longitudinal arch. Top holes do not need to be laced. Use of government issue foot powder, massage, manipulation, contrast baths, exercises, wearing two pairs of socks and cutting toenails straight across are helpful in relieving and preventing foot symptoms.

TABLE 2.—*Degree of Pronation*

	Number	Pes Cavus	No Pronation or Very Slight	Moderate or 2d Degree	Pronounced or 3d Degree
Symptomatic group receiving medical discharge.....	75	3	0	32	40
Nonsymptomatic group.....	347	0	288	52	7

TABLE 3.—*Occupation*

	Number	Occupation		Age		
		Sedentary	Active	Under 20	20-30	Over 30
Symptomatic group....	75	27 (36%)	47 (62%)	2	67	5
Nonsymptomatic group	141	62 (44%)	79 (56%)	2	106	33

SUMMARY

1. Foot complaints constitute one of the major problems in military medicine.

2. Pronounced pronation of the foot may be present without symptoms. Two per cent of a series of "normal" functioning feet presented third degree pronation and 15 per cent second degree pronation.

3. A light, flexible, adjustable leather arch support can be easily made in any army hospital.

Intelligence.—Education generally acknowledges that ability to act intelligently depends on native endowment, growth and opportunity for experiences of educational potentialities. Intelligence does not remain static. Improvement in environment will bring about a gain in intelligence. In the ultimate outcome, inheritant capacities make more contributions than environment. "Some authorities claim that from 75 to 80 per cent of the intelligence quotient variance is due to innate and heritable causes while about 17 per cent is due to difference in home environment and that home environment in rare, extreme cases may account for as much as twenty points of increment above the expective or congenital level." Bingham states: "The evidence at present in hand indicates that there is about one chance in twenty-two that during a four year period intelligence quotient will either increase or decrease as much as this, and that about one chance in three that it will change as much as six points."—Davis, John E.: Principles and Practice of Rehabilitation, New York, A. S. Barnes & Co., Inc., 1943.

PAINFUL FEET

CONGENITAL INSUFFICIENCY OF THE FIRST METATARSAL SEGMENT AS A CAUSE AMONG SOLDIERS RECENTLY INDUCTED INTO THE ARMY

CAPTAIN ROBERT BINGHAM
MEDICAL CORPS, ARMY OF THE UNITED STATES

A surprisingly large number of soldiers who have been recently inducted into the Army develop the complaint of "painful feet" during the period of their basic training. Fortunately, most of these symptoms are due to minor functional strains from the physical conditioning program. These respond very well to the treatment of the battalion or dispensary surgeon with rest and conservative management. The more severe cases are referred to the Orthopedic Clinic, and in a large training camp hospital they can be divided into two groups: (1) traumatic lesions of the feet, severe strains, sprains and fractures, and (2) postural foot disorders and skeletal abnormalities. This second group generally requires the most careful diagnosis and prompt treatment if men thus affected are to be salvaged for some useful type of military service.

THE FIRST METATARSAL SEGMENT

A frequently unrecognized and undiagnosed cause of foot pain in the new soldier is a congenital insufficiency of function of the first metatarsal segment. Slightly over 10 per cent of all patients with foot disorders of the second class were found to have this pathologic condition during a six month observation period at the Orthopedic Section of the Surgical Service, Station Hospital, Camp Van Dorn, Mississippi. Its medical importance and military significance both lie in the fact that with correct early diagnosis and specifically designed treatment almost all these patients can continue on full field duty. This anatomic variation in the foot has been called "Morton's syndrome" for its discoverer, Dr. Dudley J. Morton,[1] professor of anatomy of the Columbia University College of Physicians and Surgeons, New York. His lifetime of study of the pathologic anatomy of foot disorders and his original methods of diagnosis and treatment are among the greatest contributions of this generation to our knowledge of the structure and functions of the feet. Other names given to conditions arising from this syndrome are "metatarsus primus brevus varus," "Morton's principle" and "short first metatarsal." In addition there are many types of foot abnormalities which some orthopedic surgeons believe originate in this syndrome—some of which are misnomers—"fallen metatarsal arches," "painful transverse arches," metatarsalgia, weak feet, low longitudinal arches, flat feet and pronated feet. Indeed, whenever one of the aforementioned diagnoses has been made in civilian life and the treatment received has been unsuccessful in relieving the symptoms, it has been a common occurrence on clinical examination to find a case of short or relaxed first metatarsal segment present which has been symptomatic but not previously recognized or adequately treated.

The syndrome of shortness of the first metatarsal segment should not be confused with the more rare

From the Orthopedic Section, Surgical Service, Thirty-First General Hospital.

1. Morton, Dudley J.: The Human Foot, New York, Columbia University Press, 1935: Foot Disorders in General Practice, J. A. M. A. **109**: 1112-1119 (Oct. 2) 1937; Functional Disorders of the Feet and Their Treatment, New York State J. Med. **42**: 2119-2123 (Nov. 15) 1942.

pathologic lesion known as "Morton's sore toe" or "Morton's metatarsalgia," which was first described by Professor T. G. Morton in 1876. Both conditions may be responsible for a clinical metatarsalgia, but in the latter condition the cause is considered to be a definite neuroma of one of the plantar nerves. In my experience the incidence of these cases is very small compared with the number of patients who have metatarsal pain from short first metatarsal segment, and I have not found it necessary to resort to surgery of the plantar nerves to effect a clinical cure.

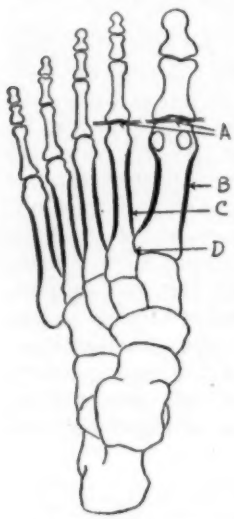


Fig. 1.—Diagram of the x-ray features of normal foot structure. *A*, first and second metatarsal shafts are equal in length; *B*, thicker cortex; *C*, thin cortex; *D*, normal joint space.

The principal anatomic or functional insufficiency of the first metatarsal segment of the foot is the underlying cause for the production of the symptoms (fig. 1). This is due to a congenital developmental defect of the first metatarsal shaft, which may be either (1) shortened, so that it cannot bear its share of the weight thrust from the sole of the foot (fig. 2); (2) relaxed, so that it is dorsally hypermobile and can be displaced upward, causing a functional shortening (fig. 3); (3) abducted to a varus position out of the weight bearing line of the longitudinal arch (fig. 4); (4) functionally shortened by posteriorly displaced sesamoids, or (5) failing to bear a proper share of the weight of the body, owing to a combination of these deformities (fig. 5).

The condition is usually bilateral, and symptoms and changes in foot structure vary with the severity and the duration of the syndrome. The entire range of foot abnormalities which may result from this condition have been completely described and their cause determined in the publications of Dr. Morton, but a clinical study of a large number of these patients has not previously been published.

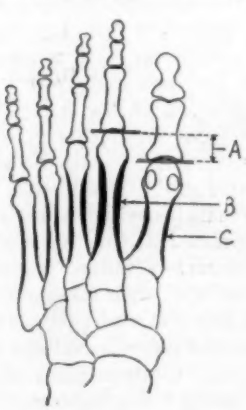


Figure 2.

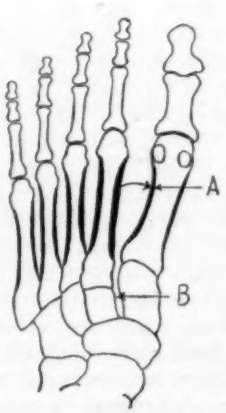


Figure 3.

Fig. 2.—Shortening of the first metatarsal shaft, with hypertrophy of the cortex of the second metatarsal shaft, the chief characterisic of Morton's syndrome, as diagramed from typical x-ray findings. *A*, shortness of first metatarsal segment; *B*, thicker cortex of the second metatarsal shaft; *C*, thinner cortex of the first metatarsal shaft.

Fig. 3.—Laxity of the first metatarsal shaft, resulting in widening of the joint space between the first and second cuneiforms. *A*, laxity of the first metatarsal shaft; *B*, widening of the joint space.

DIAGNOSIS

For clinical and practical purposes it is necessary to know only the primary and fundamental changes in foot structure and function which result from the instability of the first metatarsal segment. These are the following:

1. The second metatarsal segment bears an increased amount of the body weight, resulting in enlargement of the shaft, thickening of the cortex, painful pressure under the second metatarsal head and strain and aching in the midtarsal joints.

2. Callus formation develops under the distal head of the second metatarsal and to a less extent at the third and fourth.

3. There is a tendency of the foot to pronation, with lowering of the longitudinal arch.

SYMPTOMS

Aching and burning pain under the callus at the region of the second and third metatarsal heads was the most common presenting symptom in this series of patients. Many had pain and stiffness in the second and third metatarsophalangeal joints described as being "behind the toes," "under the front of the foot" and "inside the middle of the foot." Longitudinal arch pain, swelling and redness at the midtarsal region and aching of the calf, hamstring and back muscles were quite frequent. These symptoms were characteristically functional and dynamic and were relieved completely by rest and aggravated by a long period of standing, marching or drilling. With the pain was frequently excessive foot fatigue.

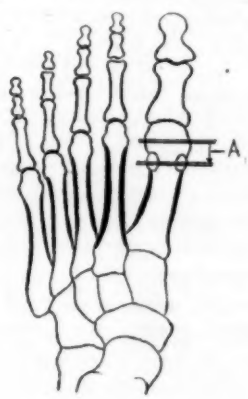


Figure 4.

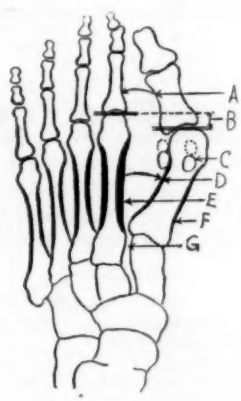


Figure 5.

Fig. 4.—Posterior displacement of the sesamoids, with resulting functional shortening of the first metatarsal segment and second metatarsal hypertrophy. *A*, displacement of the sesamoids posteriorly.

Fig. 5.—Combination of all three factors in a severe case of Morton's syndrome. *A*, hallux valgus; *B*, short first metatarsal shaft; *C*, displaced sesamoids; *D*, metatarsus varus; *E*, thickened cortex; *F*, thinner cortex; *G*, widened joint space and laxity of the joint.

DURATION OF SYMPTOMS

Acute symptoms were seldom of more than a few weeks duration, but most patients stated that their feet had "always been bad" or "too weak to do anything." In every instance close questioning revealed that the patients had been conscious of some foot weakness or abnormality in civilian life (E. P. T. E., existing prior to entrance into the Army). Some had tried arch supports or metatarsal pads without much benefit. But most of them found that their symptoms were being aggravated by their new and strenuous military duties

and were seeking medical consultation for the first time. This was particularly true of persons who followed sedentary occupations or trades while in civilian status.

PHYSICAL FINDINGS

On casual inspection many of these patients have feet that appear very nearly normal, and a careful physical examination is indicated to avoid the mistake of missing this syndrome and labeling the soldier a "gold brick." The symptoms are frequently quite pronounced in contrast to the few physical signs. For this reason an x-ray check is advisable in every case in which this pathologic condition is suspected.

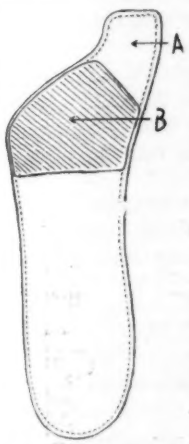


Fig. 6.—Diagram of a compensating insole. *A*, platform raised beneath the first metatarsal head; *B*, optional metatarsal pad or transverse bar.

The deficiency of the first metatarsal segment is determined by palpation of the first metatarsal shaft. It is shortened and may be slightly displaced upward and medially by pressure. With the sole of the patient's foot raised toward the examiner, he should palpate the metatarsal heads, pressing upward on the ball of the foot similar to the pressure incurred in standing. The heads of the second and usually the third metatarsals are found to be unusually prominent while the head of the first is depressed, short, abnormally moveable and covered with smooth, soft, non-weight bearing skin. This shortness, relaxation and hypermobility may be further exaggerated by a posterior displacement of the weight bearing sesamoid bones, which causes a further increase of symptoms. Sesamoiditis, however, is rare except in the extremely pronated foot.

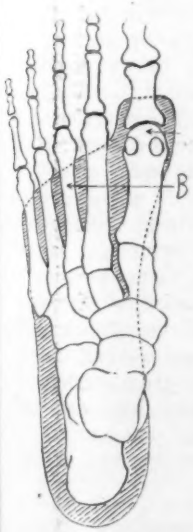


Fig. 7.—Compensating insole diagramed in place to show support of the first metatarsal segment. *A*, platform under the first metatarsal head; *B*, cushioning behind the second, third, fourth and fifth metatarsal heads.

Callus formation may be localized in the middle of the forefoot or may be discrete and under separate metatarsal heads from the second to the fifth. In some instances the great toe becomes weight bearing and a hard callus is found under the medial aspect of the interphalangeal joint. A third less common site for callus is in the "bunion" position on the medial side of the distal head of the first metatarsal.

Acute symptoms are most often due to a traumatic synovitis of the second and third metatarsophalangeal joints and the middle cuneiform joints. There may also be spasm in the toe extensor muscles with temporary dorsiflexion of the toes and later contractures and claw toes resembling hammer toes.

Chronic symptoms come from secondary hypertrophic osteoarthritis. The usual sites are the first metatarsophalangeal joint, when the great toe has been plantar flexed for compensatory weight bearing, and the cuneiform joints. These joints may be enlarged and tender, but confirmation of this complication should be obtained by x-ray examination.

The inadequate support of the short and relaxed first metatarsal segment weakens the medial border of the longitudinal arch of the foot. This causes pronation, found in many of these cases, as the foot rolls inward and downward to secure some support from the first metatarsal shaft. Flattening of the longitudinal arch occurs gradually as the result of absence of support from the short and relaxed first metatarsal segment and stretching of the ligaments in the tarsal region.

Secondary symptoms of this syndrome are therefore similar to the symptoms of weak feet, pronated feet and flat feet but are usually less severe and the physical findings less pronounced. Other foot abnormalities, pes cavus, club foot, congenital pes planus hallux valgus, bunion, hammer toe, contracted calf muscles and plantar warts were found associated with this syndrome occasionally but seem to have no etiologic relationship.

TABLE 1.—*Analysis of Cases*

I.	**The incidence of patients with Morton's syndrome:**		
	Orthopedic patients seen in six months from a group of 10,000 soldiers in training	650	6.50%
	Patients with "painful feet"	332	3.32%
	Patients with Morton's syndrome	34	0.34%
II.	**Typical findings in 100 patients with Morton's syndrome:**		
	1. Symptoms: Acute	18	
	Subacute	46	
	Chronic	36	
	Metatarsalgia	90	
	Callus under 2d and 3d metatarsal heads	76	
	Longitudinal arch pains	62	
	Excessive foot fatigue	72	
	Muscular pain in legs	18	
	Backache	12	
	2. Duration of symptoms:		
	a. Longest	17	years
	Shortest	1	week
	Average duration	2	years
	b. Age at which symptoms appeared:		
	Earliest	11	years
	Oldest	38	years
	3. Physical findings:		
	Short 1st metatarsal shaft	90	
	Dorsal hypermobility	32	
	1st metatarsus varus	18	
	Displaced sesamoids	4	
	4. Secondary findings:		
	Pronated feet	16	
	Pes planus	26	
	Weak feet	18	
III.	**Treatment prescribed for 100 patients:**		
	1. Morton's compensating insoles	90	
	2. Exercises for strengthening feet	18	
	3. Hydrotherapy	14	
	4. Duration of therapy:		
	Treated in civilian life by other methods	24	
	Symptoms but no treatment in civilian life	10	
	First symptoms and treatment in Army	66	
	Number treated as outpatients	96	
	Number admitted to the hospital	4	
	Average duration of treatment	8	days
	Minimum treatment	2	days
	Maximum treatment	21	days

TABLE 2.—*Results of Treatment*

1.	Soldiers returned to full military duty (general service)	76
2.	Soldiers reclassified for noncombatant duty (limited service)	14
3.	Patients under treatment or awaiting reclassification	10

X-RAY FINDINGS

The most valuable and accurate means of confirming the diagnosis of Morton's syndrome is an anteroposterior film of the foot in the standing position centered over the second cuneiform bone (dorsoplantar x-ray view). The findings are pathognomonic regardless of the severity or mildness of the symptoms in the particular case (figs. 2, 3, 4 and 5). The first meta-

tarsal is shortened, compared to the second, and is very frequently in varus. The cortex is more thin than normal and may appear atrophic. Osteoarthritis of the first metatarsophalangeal joint is an occasional finding. The sesamoids may appear displaced posteriorly or adducted. A lateral view may show a relative dorsiflexion of the first metatarsal shaft. Relaxation is further evidenced by widening of the joint between the first and second metatarsals and the first and second cuneiforms. The second metatarsal shaft is hypertrophied and enlarged, the cortex is thickened and there is evidence of increased functional stress. The third and fourth metatarsal shafts may show similar changes to a less degree.

These x-ray findings verify the specific skeletal changes which occur as congenital abnormalities and as developmental functional lesions in this disorder and prove its pathologic entity. This fact, together with the specificity of its treatment, is sufficient that this condition should be classed and described as a separate syndrome in the terminology of foot conditions.

TREATMENT

The original and effective principle of treatment has been demonstrated by Dr. Morton to be the restoration of the weight bearing function and normal position of the first metatarsal segment of the foot. This is most simply done by placing a platform or support of the proper thickness under the sole of the foot beneath the distal head of the first metatarsal. The specific treatment compensates both for the shortness and for the relaxation of the first metatarsal segment, gives support to the medial side of the longitudinal arch and relieves the symptoms caused by excessive weight thrust on the second metatarsal and midtarsal region of the foot.

In its most practical form this treatment consists in prescribing a "compensating insole," which is fitted by shoe size and has a built-in platform which extends out under the head of the first metatarsal pad or bar. The platform at the base of the great toe can be raised or lowered as needed.

This device can be made from a suitable thickness of sole leather from a tracing of the patient's foot. This can be done by any shoemaker or a soldier with special training. A low cost commercial model is also available which has superior wearing qualities and a sponge rubber metatarsal bar and can be obtained on prescription of an orthopedic surgeon or medical officer.

After the insoles are supplied to the patient they are checked for fitting and the proper height of the platform. Then they are worn for an hour or more daily, the time being increased until they are comfortable and the symptoms from the disorder have disappeared. The patient transfers them from one pair of shoes to another in changing footwear, never going without them again for more than a few hours. A soldier accustomed to their use would no more go without them on a march than he would study without glasses if necessary for reading.

General treatment is emphasized for the first week or so for the relief of acute symptoms and to strengthen the foot muscles whenever necessary. Exercises, contrast and whirlpool baths, and periods of rest and elevation of the feet are used. If the soldier is being treated as an outpatient, a period of seven to ten days on "light or fatigue and kitchen police duty" is often recommended to his company commander.

The results of this treatment and a comparison of the findings in a group of 100 patients is shown in the tables.

SUMMARY

1. Ten per cent of the soldier patients in the Orthopedic Clinic of the Station Hospital at Camp Van Dorn, Mississippi, who complained of "painful feet" were found to have "Morton's syndrome," or congenital insufficiency of the first metatarsal segment.

2. This foot disorder is a clinical entity characterized by metatarsalgia and midtarsal pain caused by a congenital developmental shortening or relaxation of the first metatarsal segment of the foot.

3. Diagnosis can be made by physical examination and verified by x-ray films in every case. Treatment consists in supplying an individually fitted "compensating insole" which provides a weight bearing platform for the first metatarsal head.

4. Of 100 soldiers treated by this method, 76 have been able to continue full military duty, "general service," while wearing the "compensating insoles."

ACUTE PORPHYRIA

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Acute porphyria is a disease which has been recognized for many years and which has received considerable attention in the literature but which has been neglected in medical teaching, so that it is totally unfamiliar to many or most physicians. I [1] have made the suggestion that this disease is not as unusual as was formerly supposed and that with a more general knowledge of the subject more cases will be detected. My purpose in the present communication is to review the clinical aspects of the disease and to present 3 additional cases of unusual interest occurring in the same family which have been encountered during the past year.

For a proper understanding of the disease, certain historical aspects of the problem are worth noting. In 1841 Scherer [2] described a red pigment formed by the action of strong sulfuric acid on hemoglobin. Although similar observations were made by Mulder,[3] an adequate study of this substance was not undertaken until 1871, when Hoppe-Seyler described certain of its chemical and physical properties and named it hematoporphyrin. Baumstark [4] aroused an interest in the part hematoporphyrin might play in medicine when he discovered similar pigments in the urine of a patient who had leprosy, and by 1892, when Garrod reviewed the subject,[5] it had been demonstrated that "hematoporphyrin" occurred in the urine of healthy persons in small amounts and that larger amounts were present in the urine of patients who had a great variety of diseases. Günther [6] described patients who excreted so much red pigment that the urine was colored, a condition which he was convinced constituted a clinical

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1. Nesbitt, Samuel, and Watkins, C. H.: Acute Porphyria, Am. J. M. Sc. **203**: 74-83 (Jan.) 1942.
2. Günther, H.: Die Hämatoporphyrie, Deutsches Arch. f. klin. Med. **105**: 89-146 (Dec. 20) 1911.
3. Mason, V. R.; Courville, C., and Ziskind, E.: Porphyrin in Human Disease, Medicine **12**: 355-439 (Dec.) 1933.
4. Baumstark, F.: Zwei pathologische Harnfarbstoffe, Arch. f. d. ges. Physiol. **9**: 568-584, 1874.
5. Garrod, A. E.: On the Occurrence and Detection of Haematoporphyrin in the Urine, J. Physiol. **13**: 598-620, 1892; Hematoporphyrin in Normal Urine, J. Physiol. **17**: 349-352, 1894.
6. Günther (footnotes 2 and 45).

entity, which he believed to be caused by a constitutional anomaly of pigment metabolism and which he called hematoporphyria. In 1924 Fischer [7] demonstrated that patients with this disease excrete coproporphyrin and uroporphyrin but that they never excrete hematoporphyrin, which is a laboratory product and never occurs in nature. For this reason hematoporphyria is a misnomer and the term porphyria is to be preferred to the use of either hematoporphyria or hematoporphyrinuria, which terms are still occasionally encountered because of long usage. The term porphyrinuria should be reserved to indicate those instances in which the porphyrins occurring naturally in the urine are present in amounts above the normal range, a condition which exists in a great variety of diseases.

The porphyrias have been classified as congenital porphyria, acute porphyria and chronic porphyria. Congenital porphyria is a disease possibly inherited as a mendelian recessive which is characterized clinically by its appearance early in life or at birth, the excretion of large amounts of porphyrin, particularly uroporphyrin I, in the urine so that the color of the urine may be dark red at times, photosensitivity, predominance in males and discoloration of the teeth by impregnated uroporphyrin. It is an extremely chronic disease the outstanding feature of which is blistering, scar formation and eventual deformity of the exposed parts as a result of the light sensitivity. In 1938 Turner and Obermayer [8] discussed 86 cases, 9 of which were considered doubtful. Acute porphyria differs fundamentally from congenital porphyria in that it is possibly inherited as a mendelian dominant, appears later in life, usually during the third and fourth decades, occurs in females more frequently than males, the ratio being about 3:1, photosensitivity is rare, pigmentation of the teeth is absent, uroporphyrin III is almost always predominant in the urine as a metal complex along with other pigments, and clinically the disease is first indicated by complaints referable to the abdomen or central nervous system. Acute porphyria has been further classified as acute toxic porphyria and acute idiopathic porphyria.[9] It has been pointed out [10] that the acute toxic forms and the acute idiopathic forms of porphyria are indistinguishable either clinically or by laboratory methods, the acute toxic form being recognized only on the basis of the presence of a toxic agent which apparently is an etiologic factor. Acute porphyria has also been classified [11] on the basis of the clinical picture as the classic type with abdominal and nervous manifestations, the abdominal type with abdominal colic only, the purely nervous type with central nervous manifestations without abdominal colic, the comatose type in which severe central nervous system involvement with coma is the outstanding event, and the latent type, which is asymptomatic despite the fact that the patient presents laboratory evidence of the fundamental pigment disorder. These are merely descriptive terms, and at different times a patient may fit into any one of these various type pictures. Up to 1939 over 250 cases of acute porphyria had been reported.[12] A number of cases have been reported more recently.[13] Chronic porphyria includes a group of cases characterized by a pronounced increase of porphyrin excretion and slight photosensitivity, as well as symptoms referable to the intestinal tract, which could not be classified as either congenital or acute porphyria. It is possible that these cases do not constitute a separate group but in reality represent either congenital or acute porphyria in mild form. The present communication will be restricted to a discussion of acute porphyria, and the reader is referred to more appropriate reviews concerning the other forms of porphyria.[14]

Acute porphyria is in reality a chronic condition [15] characterized usually by exacerbations and remissions over a period of time ranging from months to as long a period as twenty-five years. A history suggestive of familial occurrence may often be obtained. In the past the patient may have suffered from time to time from any of an assortment of ill defined complaints including vague, poorly localized abdominal pain, bizarre pains in the extremities or elsewhere, nervousness, insomnia, depression and generalized or localized weakness; the urine occasionally may have been noted to be of an unusual dark or red color at times. It may be noted, perhaps because of these earlier complaints, that the patient has been taking certain medications, or he may have been otherwise exposed to certain toxic agents which include lead, acetanilid, nitrobenzol, sulfonal, trional, various barbiturates and perhaps sulfanilamide.[16] The acute episode [17] is characterized chiefly by gastrointestinal disturbances and involvement of the central nervous system. Commonly there is severe cramplike abdominal pain, which may be generalized or which may be confined to the lower abdomen or about the umbilicus. There is associated usually nausea, vomiting and constipation, which may amount to complete ileus. There may or may not be fever or leukocytosis. Roentgenographic examination may reveal dilatation of some segment of the gastrointestinal tract, usually the duodenum, terminal ileum or a segment of the colon.[18] Jaundice may occur, and liver damage may be demonstrated by appropriate laboratory tests.[1] Involvement of the central nervous system may be manifested by symptoms ranging from paresthesias and pain in the extremities to paralysis, delirium with hallucinations of vision and hearing, epileptic seizures and coma.[19] The classic type of neurologic involvement has been described as that of

7. Fischer, Hans, and Zerweck, W.: Zur Kenntnis der naturlichen Porphyrine: V. Ueber Koproporphyrin im Harn und Serum unter normalen und pathologischen Bedingungen, Ztschr. f. physiol. Chem. **132**: 12-33, 1924.

8. Turner, W. J., and Obermayer, M. E.: Studies on Porphyria: II. A Case of Porphyria Accompanied with Epidermolysis Bullosa, Hypertrichosis and Melanosis, Arch. Dermat. & Syph. **37**: 549-572 (April) 1938.

9. Schie, E.: Acute Porphyria Without Porphyrinuria, Acta chir. Scandinav. **82**: 618-626, 1939. Mason, Courville and Ziskind.[3] Watson.[46]

10. Nesbitt and Watkins.[1] Mason, Courville and Ziskind.[3] Watson.[46]

11. Waldenström, Jan: Studies on Porphyria, Acta med. Scandinav., 1937, suppl. 82, pp. 1-254.

12. Chandler, F. G.; Harrison, G. A., and Rimington, C.: Clinical Porphyrinuria with Report of a Case of the Acute Idiopathic Type, Brit. M. J. **2**: 1173-1180, 1939. Waldenström (footnotes 11 and 20).

13. Geissler, J.: Zur Kenntnis der Porphyria acuta, Klin. Wchnschr. **18**: 378-380, 1939. Johanning, K.: Acute Hemolysis and Porphyria in Pregnancy: Case, Nord. med. (Med. Rev., Bergen) **2**: 1763-1766, 1939. Lüthy, F.: Ueber die Erblichkeit und Therapie der Porphyrie, Klin. Wchnschr. **18**: 1034, 1939. Vahlquist, B.: Die quantitative Bestimmung des Porphobilinogens im Harn von Kranken mit sogenannter akuter Porphyrie, Ztschr. f. physiol. Chem. **259**: 213-221, 1939. Wiig, N.: Porphyrins and Their Significance in Pathology, with Special Regard to Acute Abdominal Porphyria, Nord. med. (Med. rev., Bergen) **8**: 1963-1967 (Nov. 9) 1940. Rau, Leo: Acute Idiopathic Porphyria, Lancet **2**: 647-648 (Nov. 23) 1940. Ford, W. H., and Ulrich, H. L.: Acute Porphyria, Minnesota Med. **24**: 254 (April) 1941. Nesbitt and Watkins.[1] Chandler, Harrison and Rimington.[12] Palmer.[62] Watson and Schwartz.[27] Eliaser and Kondo.[58] Correll, Peters and Murphy.[21] Hoagland.[19]

14. Dobriner, K., and Rhoads, C. P.: The Porphyrins in Health and Disease, Physiol. Rev. **20**: 416-468 (July) 1940. Mason, Courville and Ziskind.[3] Günther.[45] Watson.[46] Stokes, Beerman and Ingraham.[57]

15. Waldenström.[11] Turner.[59]

16. Taylor, F. R.: Porphyria, Urol. & Cutan. Rev. **46**: 104-108 (Feb.) 1942. Nesbitt and Watkins.[1] Mason, Courville and Ziskind.[3] Palmer.[62] Waldenström.[20]

17. Nesbitt and Watkins.[1] Mason, Courville and Ziskind.[3] Watson.[46]

18. Mason, Courville and Ziskind.[3] Palmer.[62] Turner.[59]

19. Hoagland, P. I.: Acute Porphyria: Report of Two Cases with Neurologic Manifestations, Proc. Staff Meet., Mayo Clin. **17**: 273-280 (May 6) 1942. Nesbitt and Watkins.[1] Mason, Courville and Ziskind.[3] Watson.[46] Waldenström (footnotes 11 and 20). Palmer.[62]

an ascending, Landry's type of paralysis. Of 143 cases described by Waldenström,[20] 60 showed neurologic symptoms. In these instances most of the pareses were irregularly distributed, sometimes involving only a small muscle group, sometimes practically all the striated muscles of the body. There may be ptosis, facial palsy, diplopia, dysphonia, dysphagia and dysmasesia. Respiratory failure due to bulbar palsy is a frequent cause of death. The pareses may appear suddenly or over the course of weeks. Psychotic behavior, usually of a type called toxic psychosis or manic-depressive, may be a feature of the disease, which is usually accompanied by progressive paralysis. Menstrual disturbances, particularly amenorrhea, are frequently associated with the acute episode.[21] Photosensitivity occurs rarely and is comparatively mild.[1] There is occasionally a diffuse or spotted pigmentation of the skin, but not of the mucosa, the nature of which is unknown. The cardiovascular system is frequently involved, and hypertension is often an integral part of the picture.[22] Also transient electrocardiographic changes during the acute episode have been described consisting of an elevated ST segment in lead 1 and slurring of the ST segment in lead 3.

The diagnosis of acute porphyria depends on an evaluation of the clinical picture and must be confirmed by an examination of the urine. The diagnosis will be made only if it is borne in mind and considered as a possibility in any case of unusual abdominal complaints or symptoms referable to the central nervous system, especially when these occur together, or when associated with the excretion of abnormally colored urine. It has been considered necessary to demonstrate the presence of uroporphyrin in the urine of such patients in order to establish the diagnosis of acute porphyria.[23] Several such cases, however, have been described in which uroporphyrin was not excreted in the urine,[24] and Waldenström [20] has described such a patient who later, during a period of remission, did excrete uroporphyrin. For a discussion of the chemistry of the porphyrins the reader is referred to more appropriate reviews.[25] Almost always patients with acute porphyria excrete uroporphyrins III and I in the urine, with a great predominance of the type III isomer, although several cases have been reported [26] in which the type I isomer was predominant. It has been shown during recent years that much of the uroporphyrin may be excreted as the zinc metal complex,[27] and between the acute episodes that fraction which is present as the metal complex may be increased. It is important to note that the absorption bands of the zinc metal complex of uroporphyrin are almost identical with those of oxyhemoglobin; a reversion spectroscope is necessary for precise wavelength measurement, and final differentiation is facilitated by the fact that when a strong mineral acid is added the metal complex is broken up and the absorption bands of the complex are replaced promptly by those of uroporphyrin. The normally occurring coproporphyrin of the urine is usually increased above the normal range, although not always, and when so increased the isomeric type III again usually predominates. Coproporphyrin of both isomeric types I and III has been isolated from the feces,[28] but only recently has uroporphyrin been isolated from the feces of patients with acute porphyria.[29] It has been known that the urine of these patients may be perfectly normal in appearance and also that a substance is present in the urine which produces a positive Ehrlich's aldehyde reaction.[30] Waldenström and his associates [31] have described a colorless chromogen substance in the urine of these patients which they have termed porphobilinogen, which gives a strong red color with Ehrlich's aldehyde reagent and which on standing or being acidified and heated is converted partly into a red pigment resembling urobilin, called porphobilin, and partly into uroporphyrin. Watson and Schwartz [32] have devised a simple test for urinary porphobilinogen which they believe to be pathognomonic of porphyria. It is becoming evident that many pigments may be involved in this metabolic disorder. Pigments other than porphyrins may account for most or all of the dark color of the urine of these patients, which may range from colorless or normal yellow to red or black, depending on the relative amounts of the various pigments which it may contain. The chemical nature of most of these is little understood. Included among them are melanin,[33] skatole red,[34] indole, leucine, tyrosine and cystine [35] and urofuscin.[36]

It is obvious that the differential diagnosis of acute porphyria involves many possibilities. Almost always the acute episode is characterized by severe abdominal pain, nausea and vomiting, and constipation which may amount to complete ileus; there may be fever and even leukocytosis as high as 20,000 cells per cubic millimeter of blood, or there may be menstrual disturbances, notably amenorrhea. The problem may therefore appear to be a surgical one, and it is not surprising to know that many such patients have been operated on with the mistaken diagnosis of such conditions as acute appendicitis, surgical ileus, cholecystitis, ectopic pregnancy and ovarian cyst.[22] On examination, however, the abdomen is usually soft, and it is during these

20. Waldenström, Jan.: Neurologic Symptoms Caused by So-Called Acute Porphyria, Acta psychiat. et neurol. **14:** 375-379, 1939.

21. Correll, H. L.; Peters, B. J., and Murphy, F. D.: Porphyria: A Report of Two Cases, Urol. & Cutan. Rev. **46:** 341-343 (June) 1942.

22. Waldenström (footnotes 11 and 20).

23. Mason, Courville and Ziskind.[3] Watson.[10] Waldenström.[11]

24. Backer-Gröndahl, N.: Porphyria Without Porphyrin, Acta chir. Scandinav. **76:** 227-248, 1935. Ravn, J.: Disturbances of Liver Function in Two Cases of Acute Porphyria, Acta psychiat. et neurol. **10:** 459-480, 1935. Schie.[9]

25. Waldenström, Jan: Some Observations on Acute Porphyria and Other Conditions with a Change in the Excretion of Porphyrin, Acta med. Scandinav. **83:** 281-316, 1934. Fischer, H., and Orth, H.: Die Chemie des Pyrrols, Leipzig, Akad. Verlagsgesellsch., 1937. Mason, Courville and Ziskind.[3] Watson.[10]

26. Fischer, H., and Libowitzky, H. L.: Auftreten von Uro- bzw. Koproporphyrin I bei akuter Porphyrie, Ztschr. f. physiol. Chem. **241:** 220-222, 1936. Turner.[39]

27. Watson, C. J., and Schwartz, S.: The Excretion of Zinc Uroporphyrin in Idiopathic Porphyria, J. Clin. Investigation **20:** 440-441 (July) 1941. Nesbitt and Watkins.[2] Chandler, Harrison and Rimington.[42] Turner.[39]

28. Waldenström, Jan; Fink, H., and Hoerburger, W.: Ueber ein neues bei der akuten Porphyrie regelmässig vorkommender Uroporphyrin, Ztschr. f. physiol. Chem. **233:** 1-9, 1935. Dobriner, K.: Simultaneous Excretion of Coproporphyrin I and III in a Case of Chronic Porphyria, Proc. Soc. Exper. Biol. & Med. **35:** 175-176, 1936. Mertens, E.: Ueber die bei akuter Porphyrie auftretenden Porphyrine, Ztschr. f. physiol. Chem. **250:** 57-79, 1937. Waldenström.[11] Weiss.[35]

29. Schwartz, Samuel, and Watson, C. J.: Isolation of Uroporphyrin from Feces in Idiopathic Porphyria, Proc. Soc. Exper. Biol. & Med. **47:** 390-393, 1941.

30. Turner, W. L.: Studies on Porphyria: III. Acute Idiopathic Porphyria, Arch. Int. Med. **61:** 762-773 (May) 1938. Mason, Courville and Ziskind.[3]

31. Waldenström, Jan, and Vahlquist, Bo.: Studien über die Entstehung der roten Harn pigmente (Uroporphyrin und Porphobilin) bei der akuten Porphyrie aus ihrer farblosen Vorstufe (Porphobilinogen), Ztschr. f. physiol. Chem. **260:** 189-209, 1939. Waldenström, Jan, and Wendt, S.: Tierexperimentelle Studien über den Porphyrinstoffwechsel, ibid. **259:** 157-162, 1939. Vahlquist, Bo.: Die quantitative Bestimmung des Porphobilinogens im Harn von Kranken mit sogenannter akuter Porphyrie, ibid. **259:** 213-221, 1939. Waldenström (footnotes 11 and 20).

32. Watson, C. J., and Schwartz, Samuel: A Simple Test for Urinary Porphobilinogen, Proc. Soc. Exper. Biol. & Med. **47:** 393-394, 1941.

33. van der Zoo der Jong, H. H.: Een waarshuwing biz het zoeken naar melanine en haematoporphyrine in de urine, Nederl. tijdschr. v. geneesk. **69:** 598, 1925.

34. Maase, C.: Ueber das Auftreten von Skatolfarbstoffen im Harn bei akuter Hämatoporphyrie, Ztschr. f. klin. Med. **99:** 270-285, 1924.

35. Weiss, H.: Zur Kenntnis der Porphyrinkrankheiten, Deutsches Arch. f. klin. Med. **149:** 255-278, 1925.

36. Fischer, Hans: Ueber das Urinporphyrin, Ztschr. f. physiol. Chem. **95:** 34-60, 1915.

acute phases that the urine is most likely to be darkly colored. Ordinarily by the time jaundice and hepatic damage become evident other features of the disease, either abdominal or neurologic, have been present to serve as a clue to the true nature of the disease. The blood pressure may be extremely labile, so that the simultaneous occurrence of hypotension and skin pigmentation may suggest the diagnosis of Addison's disease. Ordinarily hypertension may alternate with periodic hypotension and usually appears at some time during the disease, and the pigmentation of the skin differs from that of Addison's disease in that the mucous membrane is not involved; also the other features of acute porphyria are present to aid in the differentiation of these conditions. The neurologic manifestations of acute porphyria again may suggest a multitude of possibilities.[37] It should be noted that Landry's paralysis indicates a clinical course and is not a disease entity. Most important in the differential diagnosis of acute porphyria is infectious polyneuritis,[38] which includes the Guillain-Barré syndrome, acute febrile polyneuritis, and acute polyneuritis with facial diplegia, the clinical picture of which may correspond exactly to the neurologic picture of acute porphyria. Infectious polyneuritis, however, almost always occurs in the winter or autumn; usually there is the history of a recent previous infection including the common cold or grip, measles, varicella and pneumonia; also the total protein of the cerebrospinal fluid becomes elevated, ranging from 80 to 800 mg. per hundred cubic centimeters of fluid and the cells may be increased 0-26, usually all lymphocytes, with the "albuminocytologic dissociation." The cerebrospinal fluid in acute porphyria is usually negative except occasionally for the content of demonstrable amounts of porphyrin. Other neurologic conditions which may be suggested by the manifestations of acute porphyria include hysteria, psychoneurosis, acute psychosis, encephalitis, brain tumor, poliomyelitis, progressive muscular atrophy, familial recurrent paralysis, familial periodic paralysis, diphtheritic polyneuritis, tick paralysis,[39] triorthocresyl phosphate poisoning (jake leg and apiol paralysis), serum paralysis and parotitic paralysis. Other conditions which might be considered in the differential diagnosis of acute porphyria include periarteritis nodosa, arsenic poisoning, nicotine poisoning, trichinosis and so-called hypertensive encephalopathy.

The pathogenesis of acute porphyria remains obscure, and the metabolism of porphyrins is little understood. A porphyrin constitutes an integral part of the molecular structure of certain vital respiratory pigments, notably hemoglobin, myoglobin and cytochrome. Much evidence has accumulated which would indicate that porphyrin arises in the body during the process of hemopoiesis rather than during the destruction of hemoglobin, as had been supposed formerly.[40] Waldenström [20] reminds us that porphyria might also represent a severe disorder in the metabolism of myoglobin. Turner [41] suggests the possibility that in porphyria cells other than those of the erythropoietic system may be active in porphyrinogenesis, such as those of the liver, and Turner [42] has presented evidence which suggests that porphyria may represent a persistence of fetal pyrrole metabolism. A possibility has been mentioned that the metal complex of uroporphyrin may be formed in the intestinal tract or that it may be formed in the body and excreted by the intestinal mucosa.[43] Several ingenious theories have been suggested as the possible mechanism responsible for the synthesis of porphyrin within the body which may well depend upon an enzyme system.[44] Günther [45] believed that porphyria represents an inborn error of pigment metabolism, and it is reasonable to consider that this may be due to a congenital defect in the enzyme system which may be involved in the formation of porphyrin within the body. Those reports which indicate that the porphyrias are a familial disease have done much to confirm Günther's original idea. Families have been observed in which more than one member was afflicted with the manifestations which are characteristic of acute porphyria, and in some instances there has been proof of such diagnosis by chemical means; in a few instances one member of the family suffered from acute porphyria while another member of the same family suffered from congenital porphyria.[46] Waldenström [11] described many families in which both the acute toxic and the acute idiopathic forms of porphyria occurred in the same family, and he is of the opinion that acute porphyria is inherited and transmitted as a dominant mendelian characteristic. He points out that, although the disease is almost certainly a chronic disorder of constitutional origin, the cause of the acute attack is almost totally obscure.[20] It has repeatedly been demonstrated that the metabolic error is not limited to the periods of acute symptoms in these patients but that they may excrete uroporphyrin and its metal complex between such episodes.[47] It has been mentioned previously that a variety of drugs or toxic substances have been incriminated as etiologic factors in producing acute porphyria. It had formerly been supposed that a constitutional liability need not necessarily be assumed in every instance and that acute toxic and acute idiopathic porphyrias might be caused by some endogenous or exogenous intoxication which affects certain persons who may have an idosyncrasy.[48] It might be assumed, of course, that there is always some toxic agent involved as an etiologic factor but that in cases of acute idiopathic porphyria the toxic agent remains unrecognized. Support for such an assumption might be interpreted from a report by Weiss [35] of 2 patients in whom acute idiopathic porphyria developed, 1 of the patients having replaced

37. Waldenström.[20] Hoagland.[19]

38. Roseman, E., and Aring, C. D.: Infectious Polyneuritis, Medicine **20**: 463-494 (Dec.) 1941.

39. Abbott, K. H.: Tick Paralysis: A Review, I, Proc. Staff Meet., Mayo Clin. **18**: 39-45 (Feb. 10) 1943; II, ibid. **18**: 59-64 (Feb. 24) 1943.

40. Dobriner, K.: Porphyrin Excretion in Feces in Normal and Pathological Conditions, J. Biol. Chem. **120**: 115-127, 1937. Dobriner, K., and Rhoades, C. P.: The Metabolism of Blood Pigments in Pernicious Anemia, J. Clin. Investigation **17**: 95-103, 1938. Dobriner, K.; Strain, W. H.; Localio, S. A.; Keutmann, H., and Stephens, D. I.: II. Coproporphyrin I Metabolism and Hematopoietic Activity, Proc. Soc. Exper. Biol. & Med. **36**: 755-756, 1937. Rimington, C.: Porphyrins and Their Relation to Metabolism of Blood Pigments, Proc. Roy. Soc. Med. **32**: 1268-1275, 1939. Lemberg, R.: Transformation of Haemins into Bile Pigments, Biochem. J. **29**: 1322-1336, 1935.

41. Turner, W. J.: A Theory of Porphyrinogenesis, J. Lab. & Clin. Med. **26**: 323-330 (Nov.) 1940.

42. Turner, W. J.: Studies on Porphyria: I. Observations on the Fox Squirrel, Sciurus Niger, J. Biol. Chem. **118**: 519, 1937. Turner and Obermayer.[8] Turner.[20]

43. Watson, C. J., and Schwartz, S.: The Excretion of Zinc Uroporphyrin in Idiopathic Porphyria, J. Clin. Investigation **20**: 440-441, 1941.

44. Rimington, C.: Some Cases of Congenital Porphyria in Cattle, Onderstepoort J. Vet. Sc. **7**: 567, 1936. Dobriner, K.; Strain, W. H., and Localio, S. A.: I. Quantitative Measurement of Coproporphyrin and Total Coproporphyrin I Excretion in Normals, Proc. Soc. Exper. Biol. & Med. **36**: 752-754, 1937. Turner.[41]

45. Günther, H.: Die Bedeutung der Hämatoporphyrine in Physiologie und Pathologie, Ergebn. d. allg. Path. u. path. Anat. **20**: 609-764, 1922; Günther, H.: Haematoporphyrine, in Schittenhelm, A.: Handbuch der Krankheiten des Blutes und der blutbildenden Organe, Berlin, Julius Springer, 1925, vol. 2, pp. 622-673.

46. Watson, C. J.: The Porphyrins and Their Relation to Disease: Porphyria, in Christian and MacKenzie's Oxford Medicine, New York, Oxford University Press, 1938, vol. 4, pp. 1-34.

47. Nesbitt and Watkins.[1] Waldenström.[20] Turner.[20]

48. Schulte: Ueber Hämatoporphyrinurie, Deutsches Arch. f. klin. Med. **58**: 313, 1897. Mason, Courville and Ziskind.[3]

the initial patient in the capacity of cook in a particular restaurant. The disease has never been reproduced in animals by the administration of large doses of the various toxic substances mentioned over long periods of time,[3] and also the familial character of porphyria has become more apparent, so that in all probability the toxic substances act only as precipitating factors in a person who has the chronic metabolic disorder. Eldahl believes that other precipitating factors include anxiety, exhaustion and infections.[49]

Our knowledge of the exact cause of the various symptoms of acute porphyria remains as obscure as is knowledge of the nature of the fundamental metabolic disorder. Derrien and Benoit[50] noted the excretion of zinc uroporphyrin and suspected that the loss of zinc from the body might be the most serious factor. Most workers believe that the symptoms are produced by the toxic effect of the abnormal types or amounts of porphyrin, or possibly of other pigments such as urofuscin which are present in the body. The abdominal pain may be due to the action of porphyrin on the sympathetic ganglions or on the smooth muscle of the intestinal tract.[3] Roentgenographic examination may reveal dilated portions of the intestinal tract, and at exploratory laparotomy portions of this tract have been found to be in extreme contraction.[51] Experimental evidence would indicate that the action of porphyrin on the autonomic nervous system of the intestine may well produce the abdominal symptoms; porphyrin either injected intravenously or applied locally interrupts the normal rhythmic contractions which the application of atropine does not restore.[52] Meissner's plexus may be inhibited by porphyrin.[53] These reactions may be scattered over the bowel so that spasm and atony may occur in the same portion of the intestinal tract. The central nervous system manifestations may well be attributed to the toxic effect of uroporphyrin or coproporphyrin, which may be present in the cells of this system either in solution or in a finely divided state.[54] That porphyrins may have an extremely toxic effect on the central nervous system has been demonstrated by the fact that a patient taking hematoporphyrin (Photodyne) developed a severe multiple peripheral neuritis.[55] The cardiovascular manifestations, notably hypertension, may be due to arteriospasm. Waldenström[20] has seen angiospasm of the retinal arteries during transient amaurosis, and changes have been noted in the electrocardiogram of a patient with acute porphyria which were interpreted as due to transient myocardial ischemia rather than to infarction, and evidently resulting from coronary artery spasm.[56] In experimental studies following the injection of coproporphyrin or uroporphyrin into rabbits, the electrocardiogram showed some transient changes.[53] Photosensitivity in acute porphyria is rare, but when it does occur it is doubtless caused by porphyrins, which are well known for their photodynamic properties.[57] Porphyrins of isomeric series III have less photodynamic action than do those of isomeric series I. Because of the fact that patients with acute porphyria excrete chiefly type III porphyrin while those patients who have congenital porphyria excrete predominantly type I porphyrins, it might be supposed that this accounts for the fact that photosensitivity is the feature of the latter disease but is rare in the former. However, several patients with acute porphyria have been described who excreted chiefly type I porphyrins yet did not exhibit photosensitivity. The frequent association of the acute exacerbation of acute porphyria with menstrual irregularities, notably amenorrhea, has led some to suspect an endocrine factor, though the significance of this is in doubt.[21] Hyperthyroidism has been reported as developing suddenly in several cases.[58] One case with a Fröhlich's type of obesity has been described.[59] Grünewald reported a case characterized by the secretion of colostrum,[60] and Harbitz reported a case occurring in a man who had swelling of the breasts during an acute episode.[61]

Pathologic findings in acute porphyria are usually disappointing. The demonstrable changes in the central nervous system are most often absent or minimal,[62] consisting of a degenerative process of the peripheral nerves and in the anterior horn cells, an outstanding feature being a patchy degeneration of the myelin sheath. Changes are described in the cells of the sympathetic ganglions including pigmentation, chromatolytic and fusion forms of tigroid changes, nuclear and intracapsular vacuolization, and localized round cell accumulations. More pronounced changes have been described[63] which have been essentially cytologic; the cortical cells showed decided pyknosis and in some instances vacuolization of nuclei; many showed no chromatin material and no nucleolus; masses of amorphous pigment were seen in all sections, both iron containing and non-iron containing pigment; the motor cells of the anterior horns of the spinal cord and to a less extent those of the posterior horns at all levels showed apparent pyknosis and shrinking, and the same types of pigment were observed; the Purkinje cells of the cerebellum showed definite signs of degeneration; in some places extensive myelin degeneration of the peripheral nerves was noted as well as round cell infiltration, and a few phagocytic cells containing brown pigment. Uroporphyrin or coproporphyrin either in solution or in a finely divided state has been demonstrated within the cells of the central nervous system.[54] Pathologic changes have been described in various other organs of the body.[64] The liver may be considerably enlarged, and the hepatic cells may show changes ranging from diffuse cloudy swelling to atrophy and areas of central necrosis. The hepatic cells may contain increased amounts of a brownish yellow pigment, and the Kupffer cells may be enormously

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62. Mason, Courville and Ziskind.[3] Waldenström.[20] Taylor.[23]
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64. Nesbitt and Watkins.[1] Mason, Courville and Ziskind.[3] Palmer.[63] Correll, Peters and Murphy.[21]

enlarged, the cytoplasm containing much iron containing as well as iron free pigment. The spleen may be increased considerably in size and show proliferation of the reticular cells and hyalin fibrinoid change in the reticular cells of the malpighian bodies. Large amorphous masses of the brownish pigment may be scattered throughout as well as being present within macrophagocytes within the sinusoids and also over the entire red pulp. The kidneys may show occasional nests of pigment granules, mostly iron free, within the endothelial cells of the glomerular capillaries and in the cytoplasm of the tubular epithelial cells, and there may be noticeable diffuse cloudy swelling of the epithelium of the tubules with complete atrophy in places. The same degenerative changes may occur also in the glomeruli. The bone marrow may appear hyperplastic, and occasional large cells laden with brown pigment may be seen. Considerable muscle atrophy may be present,[65] and the heart may show poor staining of the cross striations of fibers with areas of interstitial round cell infiltration as well as hemorrhages into interstitial spaces and fatty degeneration of muscle fibers.

The prognosis of acute porphyria during an exacerbation should be extremely guarded. It has been pointed out [15] that the disease is in reality a chronic one and that after its first appearance it may extend over a period of time ranging from months to many years. Of 100 patients who were the subject of the report by Waldenström [11] 20 died within one year following the appearance of the disease clinically, 2 lived eight years, and of 12 living patients 1 had lived for twenty-seven years. The immediate mortality during the acute episodes, however, may be very high, particularly on the onset of neurologic manifestations, when it may be as high as 80 to 90 per cent.[66] Frequent causes of death are respiratory failure resulting from bulbar palsy, secondary infection, particularly pneumonia, which may result from the patient's generally debilitated state associated frequently with bulbar weakness, and hepatic insufficiency.[10] As yet there is no specific or satisfactory treatment for acute porphyria, and symptomatic and supportive measures only are available. Obviously any of the toxic agents which have been incriminated as precipitating agents, notably the barbiturates during recent years, should be avoided; physical or mental fatigue as well as any infection, as far as possible, should be avoided. Intensive therapy with various vitamins has been suggested, but with doubtful or disappointing results. The use of liver preparations has proved to be of little or no avail.[67] Strong sunlight should be avoided, particularly in those occasional cases of acute porphyria in which photosensitivity is apparent.[1] Calcium forms insoluble salts with the porphyrins, and its use when parenterally administered has been reported to diminish the excretion of porphyrin in the urine as well as to relieve the abdominal pain.[68] In my experience such use of calcium has been disappointing. The nutritional status of the patient must be maintained, and this is particularly important with the occurrence of jaundice and severe hepatic damage; the use of a feeding formula by means of a duodenal tube may be necessary.[69] With the onset of neurologic manifestations the patient must be watched carefully for evidence of bulbar weakness. In such an event mucus should be aspirated from the throat and trachea when necessary, and access to a respirator should be assured. After the acute episode physical therapy, including regulated passive and active exercises and massage, may be of value.

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REPORT OF CASES

The cases to be presented occurred in the same family, of which certain essential data are presented in the geneological chart. Cases 2 and 3 were detected during an extensive survey of the family initiated during a study of case 1. The urine of 43 accessible members of the family was tested for porphobilinogen,[32] to which test the urine in cases 2 and 3 gave a strongly positive reaction, as a result of which these cases were more completely studied. The diagnosis of porphyria was further substantiated in all these cases by additional chemical studies, which included the isolation and identification of uroporphyrin from the urine. The remaining members of the family, in which the porphobilinogen test was negative, were not studied further. That the disease in this instance has been transmitted through the paternal line is evidenced by the fact that patient 3 is a paternal cousin of the first 2 patients. In this regard it is interesting to note that the paternal grandfather died at 49 years of an illness characterized by abdominal pain, complete quadriplegia and dementia, very possibly acute porphyria, although chemical proof of this is lacking.

CASE 1.—*History.*—A woman aged 24, a housewife of German and Italian parentage, had always enjoyed excellent health and never had observed anything unusual about the color of her urine. Prior to her present illness she had taken no medicine nor been exposed to evident toxic agents. Five and one-half months before coming to the New Haven Hospital she had experienced the onset of severe, diffuse, colicky abdominal pain associated with nausea and vomiting occurring at almost daily intervals and lasting for hours or days. She suffered from extreme constipation, whereas formerly her bowels had always been regular. Her family noted a distinct personality change: formerly always cheerful and pleasant, she became nervous, irritable and depressed. She feared insanity and wanted to die. She experienced "fits," when she would suddenly run screaming about the house. There was increasing weakness, so that within two weeks she could take no more than a few steps, and her hands were so weak that she could not use them. At this time a two months pregnancy was interrupted by a spontaneous abortion. She entered another hospital, where dilation and curettage was done, and it was noted that the urine, although red, contained only an occasional erythrocyte. At that time her blood pressure was 130 mm. of mercury systolic and 90 diastolic. Her convalescence was uneventful but not smooth, owing to her irritability and lack of cooperation. This was the patient's second pregnancy, her first having been normal throughout. Following that episode she seemed to regain some strength in her extremities, but her general condition became progressively worse. The abdominal pain recurred almost daily, associated still with nausea and vomiting, the longest interval of comparative freedom being about one week. She lost 35 pounds (16 Kg.). Amenorrhea was complete during this entire interval. There was increasing frequency and urgency of urination, especially at night; she was able to pass only small amounts of urine at a time. Her mental condition became worse, so that she was sent to her mother's home in the

69. Snell, A. M., and Butt, H. R.: Hepatic Coma: Observations Bearing on Its Nature and Treatment, Tr. A. Am. Physicians **56:** 321-329, 1941. Nesbitt and Watkins.[1]

country because of her screaming. She possessed ideas of persecution by members of the family and frequently threw herself down to pound her head against the floor. On one occasion she attempted to obtain iodine for suicidal purpose.

She was admitted to the surgical service of the New Haven Hospital five and one-half weeks after the onset of her illness. She appeared worried and anxious. The pulse rate was 136 and the temperature by mouth 98.6 F. The blood pressure was 140 mm. of mercury systolic and 100 diastolic. Despite the constant complaint of cramplike pain in the left upper quadrant, the abdomen remained soft and without muscle spasm. The reflexes were normal. The left ovary was palpable, and the thyroid gland was symmetrically and moderately enlarged and of normal consistency. The cramplike abdominal pain shifted to an area across the lower abdomen, recurring at half hour intervals leaving a residual sore, aching pain. On the second hospital day she was found comatose, legs extended, arms flexed, jaws tightly clenched and with tonic and clonic convulsive movements of the extremities. Fibrillary twitchings were noted over various muscle groups. The blood pressure was 155 systolic and 112 diastolic. Diagnostic impressions until this time had included ovarian cyst, ectopic pregnancy, anxiety state, hysteria, cerebral vascular accident including ruptured congenital aneurysm, encephalitis, idiopathic status epilepticus, expanding intracranial lesion, acute pituitary necrosis and catatonic schizophrenic stupor. The coma persisted, there was a pronounced fall in her blood pressure, and she was transferred to the medical service on the fourth hospital day. The patient was stuporous but responded and attempted to cooperate, being somewhat facetious. The pulse rate was 134 per minute, the temperature 101 F. and the blood pressure 70 systolic and 50 diastolic. The lungs were clear to auscultation and percussion. The pupils were round and reacted to light; the left pupil was widely dilated. The patient was emaciated; there was decided extensor weakness of the extremities and striking atrophy of the interosseous muscles of both hands. The deep reflexes were diminished to absent everywhere. The abdominal reflexes were absent; the plantar response was normal. Speech was difficult and somewhat slurred. There was no obvious cranial nerve weakness. The ocular fundi were not remarkable. Examination of the abdomen was negative except for evidence of a hugely dilated colon. The clinical picture suggested the diagnosis of acute porphyria. This impression was strengthened by the fact that the urine was found to be dark reddish brown, although it did not contain blood, and the diagnosis was confirmed by appropriate chemical investigations.

Laboratory Studies.—The concentration of hemoglobin was 11.2 Gm. per hundred cubic centimeters of blood; the erythrocytes numbered 4,390,000 and the leukocytes 7,200 per cubic millimeter of blood, with 17 per cent lymphocytes and 83 per cent neutrophils. A routine flocculation test for syphilis was negative, the blood group was of type A, a blood culture was negative, and the Aschheim-Zondek test was negative. The nonprotein nitrogen ranged from 42 to 20 mg. per hundred cubic centimeters of serum, the serum carbon dioxide content was 21.5 to 19.2 milliequivalents, the chlorides 65.3 to 99.6 milliequivalents. The total serum proteins were 5.17 Gm. per hundred cubic centimeters of serum, the albumin-globulin ratio being 3.31/1.86, and the blood sugar was 78 mg. per hundred cubic centimeters of blood. The icterus index ranged from 6.7 to 15 units, the van den Bergh test gave the indirect reaction, and the cephalin flocculation test was strongly positive. The cerebrospinal fluid was examined on three occasions, the pressure ranging from 135 to 240 mm. of water, the erythrocytes from 0 to 21, lymphocytes from 0 to 16 per cubic millimeter of fluid, the protein from 56 to 92.6 mg. per hundred cubic centimeters of fluid, the Pandy test 4 plus, the Wassermann test negative and the colloidal gold curve normal. No porphyrin could be demonstrated in the cerebrospinal fluid, which gave a negative porphobilinogen test. Roentgenographic examinations of the chest and skull gave negative results. Routine examination of the urine revealed no albumin, sugar, acetone or cells; the urine was acid in reaction and the specific gravity ranged from 1.013 to 1.032. The color of the freshly voided urine varied from a normal yellow or colorless to orange, burgundy red or dark brownish red. On standing the urine invariably became very dark and nearly black. All urine examined gave a strongly positive test for porphobilinogen. Spectroscopic examination of many specimens revealed the presence of uroporphyrin as the zinc metal complex. The porphyrins were isolated from the urine by an adsorption method similar to that described by Grotepass[70] but modified for the recovery of uroporphyrin. The porphyrins were further purified by chromatographic analysis and identified by determination of the melting point of the methyl ester. From a 8,500 cc. pooled urine collection there were isolated 17.875 mg. of uroporphyrin III and 4.46 mg. of coproporphyrin III.

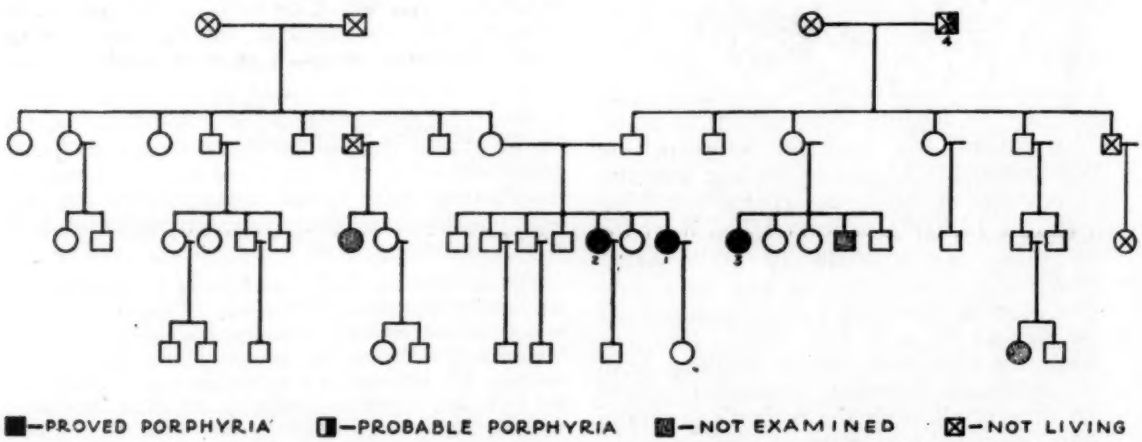

Fig. 1.—Essential data of family. Cases numbered as in text.

Treatment and Course.—Immediate treatment was directed toward the correction of salt depletion, resulting from prolonged vomiting, and vascular collapse; it consisted of transfusion and the parenteral administration of 2 per cent as well as isotonic solution of sodium chloride and 5 per cent dextrose solution. She had received large doses of barbiturates including pentobarbital sodium, phenobarbital, seconal and intravenously administered phenobarbital sodium; the use of all such medications was promptly discontinued. She received large doses of vitamin preparations administered intramuscularly, including thiamine hydrochloride about 500 mg. and nicotinamide about 600 mg. daily. The blood pressure promptly resumed previous hypertensive levels, ranging about 140 mm. of mercury systolic and 100 diastolic. Her condition was extremely variable from day to day particularly in reference to strength and consciousness. On one day there would be almost complete paralysis of her extremities, always most prominent in the proximal muscle groups, which would show considerable improvement from time to time, with increased paralysis alternating with

70. Grotepass, W.: Zur Kenntnis der natürlichen Harnporphyrine, Ztschr. f. physiol. Chem. **253**: 276-280, 1938.

periods of improvement. At times the gag reflex would be present, at other times completely absent. She steadily became weaker until there was complete flaccid paralysis of all the extremities, the deep reflexes being absent. Speech became increasingly more difficult until she could not phonate. Respiration became rapid, labored and costal in type, with an attempt to utilize the accessory muscles of respiration. There was a suggestion of hypalgesia of the first division of the fifth cranial nerve bilaterally. Swallowing became difficult, there was considerable drooling, and much watery material which became purulent was aspirated repeatedly from the trachea. The lung fields became filled with moist crepitant and crackling rales, and there was evidence of diffuse pulmonary consolidation. Despite the use of a respirator and oxygen she became more and more cyanotic, became comatose and died on the fifteenth hospital day.

Necropsy.—The only anatomic findings of importance included organizing focal pneumonia, bilateral; cystitis; ovarian cyst, left; small quiescent adenoma of the thyroid and a kyphoscoliosis of the dorsal spine. There were no changes in either the central or the peripheral nervous system. The spinal ganglions and paravertebral ganglions showed no lesions. The zinc content of the liver and pancreas was determined by the dithizone technic and was not remarkable. The liver contained 22.3 mg. of zinc per hundred grams of dry tissue, the pancreas contained 9.8 mg. A study of the porphyrin content of various organs is in progress.

CASE 2.—*History.*—A woman aged 29, sister of patient 1, a housewife, had never noted any abnormal color of her urine and at the time of examination she presented no complaints. She was attending the obstetric clinic because of a normal pregnancy of three months' duration. She had completed a normal pregnancy and delivery four years previously. Just prior to that pregnancy she had experienced a miscarriage during the fourth month; this was not associated with any unusual abdominal pain or other complaint. Five months before admission she had experienced an illness of nine days' duration characterized by severe cramplike pain across the lower abdomen, nausea and vomiting, and complete cessation of bowel movements. She was able to ingest only fluids and lost 10 pounds (4.5 Kg.). There were no neurologic symptoms. The illness was attributed by her family physician to the ingestion of warmed over succotash, although the entire family had eaten this food without ill effect. Physical examination was essentially negative. The patient was cheerful and cooperative. The blood pressure was 120 mm. of mercury systolic and 80 mm. diastolic. The heart, lungs and abdomen were normal; the uterus was enlarged symmetrically commensurate with a three months pregnancy. Examined under ultraviolet light there was no abnormal fluorescence anywhere, including the teeth and the areas of apocrine gland distribution.

Laboratory Studies.—The concentration of hemoglobin was 12 Gm. per hundred cubic centimeters of blood; the erythrocytes numbered 3,700,000 and the leukocytes 7,000 per cubic millimeter of blood. The percentages of the various types of leukocytes were neutrophils, nonsegmented 13, segmented 55, lymphocytes 26, monocytes 3 and eosinophils 2. Routine flocculation test for syphilis was negative; the blood group was type A. Routine urine examination showed a cloudy yellow color, neutral reaction, specific gravity of 1.021, 3 to 5 leukocytes per high power field, occasional epithelial cells, no albumin and no sugar. All specimens of urine examined were a normal yellow or of a colorless appearance and gave a strongly positive test for porphobilinogen. On standing the urine became extremely dark and almost black. From a 2,250 cc. forty-eight hour collection of urine there were isolated 6.755 mg. of uroporphyrin I, 1.36 mg. of uroporphyrin III and 1.81 mg. of coproporphyrin I. The native urine did not show fluorescence in ultraviolet light, nor did spectroscopic examination reveal distinct absorption.

It was evident that this patient had the fundamental metabolic disorder of porphyria, but at the time she presented no referable complaints and so was classified as having latent porphyria. It is quite possible that the illness in the past, characterized by abdominal pain, nausea, vomiting and ileus, represented an acute episode of this disease which was not recognized as such. The situation was explained to the patient and her family. She was advised never to take drugs of any kind except when absolutely necessary, then only under the direction of a physician, and in particular to avoid sedatives, soporifics and "patent medicines." She was also instructed to avoid infections as far as possible, as well as mental and physical fatigue, all of which might possibly precipitate an acute episode. The obstetric department was acquainted with the situation, and it was suggested that sedation and anesthesia be completely eliminated at the time of the patient's delivery if at all possible.

CASE 3.—*History.*—A single woman aged 26, employed as a cashier, a paternal cousin of patients 1 and 2, refused to enter for examination and her history was obtained from other members of the family. She had always enjoyed excellent health and presented no referable complaints. She had suffered from "pleurisy" for several weeks recently, which had been treated at home and the details of which were not available.

Laboratory Studies.—All urine specimens received were of a normal, clear yellowish appearance which turned nearly black on standing. The urine gave a strongly positive test for porphobilinogen. From 1,100 cc. of this patient's urine were isolated 0.82 mg. of uroporphyrin III and 0.28 mg. of coproporphyrin III.

Again it was evident that the patient had the fundamental metabolic error of porphyria which was classified as latent porphyria. This was discussed with the family, and it was stressed that she should avoid all drugs and anything which might conceivably precipitate an acute attack.

COMMENT

The problem of acute porphyria has been reviewed with particular reference to the clinical aspects of the disease, historical consideration, classification of the porphyrias, diagnosis, differential diagnosis, pathogenesis, pathology, prognosis and treatment. The terms "hematoporphyria" and "hematoporphyrinuria" are misnomers and their use should be abandoned. Acute toxic porphyria and acute idiopathic porphyria are indistinguishable clinically or by laboratory means and probably represent the same disease; acute toxic porphyria is recognized only on the basis of a toxic agent, usually a barbiturate, which may act only as a precipitating factor in causing an acute episode. Acute porphyria is in reality a chronic disease characterized usually by exacerbations and remissions over a period of months or years.

The diagnosis of acute porphyria will be made only if it is borne in mind and considered as a possibility in any case of unusual abdominal complaints or symptoms referable to the central nervous system, particularly when these occur together, or when associated with the excretion of abnormally colored urine. It would seem that the disease is not as unusual as has formerly been supposed and that with a more general knowledge of the subject more cases will be detected.

Three cases of acute porphyria occurring in the same family have been presented. The investigation of 43 members of this family was made possible by the use of a simple test for urinary porphobilinogen. In each of the 3 cases the diagnosis was further confirmed by the isolation and identification of uroporphyrin from the urine. These cases indicate that acute porphyria is as much a familial disease as is congenital porphyhia and that the underlying mechanism is an inborn error of pigment metabolism as suggested originally by Günther.

Case 1 represents the picture of classic acute porphyria and demonstrates the problems which may be presented in differential diagnosis. A feature of this patient's course was the changing neurologic picture from day to day. Necropsy findings were disappoint-

ing. The zinc content of the liver and pancreas was not abnormal and would indicate that zinc depletion is not a factor of the disease. Careful balance studies might serve to indicate what role zinc plays in this disease, which is characterized by the excretion of the zinc metal complex of uroporphyrin. It is possible that zinc may be of importance as a detoxicating mechanism, or it may be an integral factor in an as yet unknown enzyme system concerned with porphyrin metabolism.

Case 2 represents the unusual condition in which the isomeric series I of uroporphyrin and coproporphyrin are predominant in the urine; usually porphyrins of isomeric series III predominate in the urine of patients with this type of porphyria.

Cases 2 and 3 are examples of latent porphyria in which there are no clinical manifestations of the disease, although it is evident that the inborn metabolic disorder exists from the standpoint of chemical investigation. The importance of detecting such cases is indicated; the patient must be acquainted with the situation and instructed in regard to the avoidance of those things which might act to precipitate an acute episode.

THE OBLIQUELY OVATE OR NAEGELE PELVIS

A COMMENTARY AND A REPORT OF FOUR CASES

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As early as 1803 Franz Carl Naegele [1] recognized the morphology of the obliquely ovate pelvis and in his celebrated monograph published in 1839 he based his observations on the study of 37 specimens. He defined the deformity as a "pelvis contracted in one of the oblique diameters with complete ankylosis of the sacroiliac synchondrosis on one side, combined with imperfect development of the sacrum and os innominatum

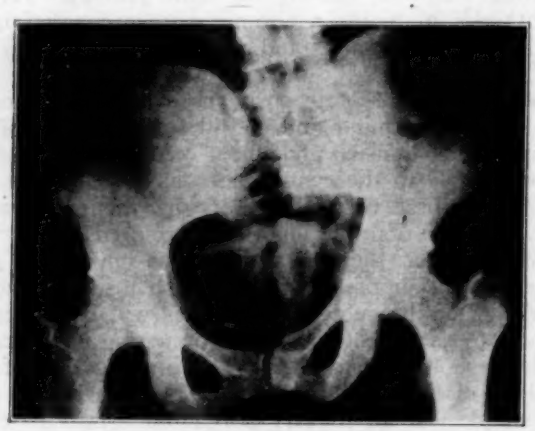

Fig. 1.—Appearance of pelvis in case 1.

on the same side." At the time of his writing, Naegele observed that the deformity had never been recognized in the living person. Today, without the aid of roentgenology, the same difficulty of diagnosis apparently obtains, as witnessed by the experience here reported.

When one considers the supposed rarity of this type of pelvic deformity, the observation of 4 cases in almost as many years in the material presenting itself in a relatively small teaching clinic is something of a revelation. In the 1941 edition of the Williams-Stander [2]

Fig. 2.—Appearance of pelvis in case 2.

textbook we read that "Thomas in 1861 was able to collect from the literature 50 of such pelves. Since then additional specimens have been described, but it is unlikely that the entire number greatly exceeds 100." Other authorities seem to be in agreement that the Naegele pelvis is one of great rarity and many obstetricians of extended experience claim never to have encountered an instance. As will be shown later, such a supposed rarity can be explained, for in some women with this type of pelvic deformity birth of a living child through the natural passages is possible.

REPORT OF CASES

A brief history of the cases that I have observed follows:

CASE 1.—A nulliparous white woman aged 33, of Irish descent, with no history of serious illness in childhood, had pyelitis in 1928 and the left kidney was removed. The specimen showed double pelvis and double ureter. In January 1938 a flat abdominal roentgenogram was made during an investigation for lower left quadrant pain. The Naegele type pelvis was seen. (Note: From 1932 to 1938 eight pelvic examinations were done by 6 men but nothing in their notes showed any evidence that the pelvic deformity was recognized.) The external appearance of the patient was not unusual; scoliosis was not readily noticeable. She never had been pregnant.

CASE 2.—An Italian woman aged 38, seen in the antepartum clinic in December 1939 in her fourth pregnancy, was first pregnant in 1925; delivery was by forceps, and the child weighed 7 pounds 8 ounces (3,400 Gm.). The second pregnancy occurred in 1928, with spontaneous delivery of a child weighing 7 pounds 14 ounces (3,570 Gm.). At the third pregnancy, in 1931, the patient was delivered at home of a child weighing "over 8 pounds" (3.6 Kg.) and was said to have suffered a birth injury. At the fourth pregnancy, vaginal examination was made by Dr. DeWitt Dominick Dec. 26, 1939, who confirmed the pregnancy and, because of abnormality of the lower pelvis, referred the patient for roentgen pelvimetry. The diagnosis was Naegele pelvis with deformity on the right side. The patient's antepartum course was uneventful until the end of the pregnancy, when symptoms of toxemia appeared and multiple pregnancy was noted. On June 18, 1940 she delivered spontaneously male twins weighing 2,830 and 2,725 Gm. (Note: This patient visited the gynecologic clinic in 1934

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1. Naegele, F. C.: Das schräg verengte Becken, nebst einem Anhange über die wichtigsten Fehler des weiblichen Beckens überhaupt, Mainz, V. von Zabern, 1839; The obliquely Contracted Pelvis, Containing Also an Appendix of the Most Important Defects of the Female Pelvis, Centennial Edition, translated by A. M. Hellman and G. Musa, New York, Pynson Printers, Inc., 1939.

2. Standar, H. J.: Williams Obstetrics, ed. 8, New York, D. Appleton-Century Company, Inc., 1941.

for "pains in the lower abdomen." At that visit and on four subsequent visits vaginal examination was done, each time by a different attendant. No mention of pelvic deformity appears on the record.)

CASE 3.—A white American woman aged 28, seen Oct. 1, 1940 in her third pregnancy, at her first visit to this clinic was examined vaginally by Dr. C. B. Crampton, who noted that the pelvis presented a deformity and referred the patient for roentgen pelvimetry. A typical Naegele pelvis was noted with deformity on the right side. The patient did not walk with any noticeable limp. The first pregnancy, in 1935, was terminated by forceps and a 7½ pound (3.4 Kg.) child delivered, now living and well. The second pregnancy, with forceps, resulted in a stillbirth of a 9½ pound (4.3 Kg.) child. The third pregnancy proceeded to term without incident and the patient was delivered by elective cesarean section of a 3,890 Gm. child. This patient gives a history of osteomyelitis during the first three years of life, which accounts for numerous scars in the region of the left hip due to incisonal drainage.

CASE 4.—An Italian woman aged 27, first seen Aug. 2, 1943, expected confinement Feb. 3, 1944. Routine roentgen pelvimetry showed a typical Naegele type pelvis. At the present time the patient is progressing satisfactorily in pregnancy. The past history is interesting for, at the age of 2 years, the patient was operated on in a local hospital for incision and drainage of the left thigh for a tuberculous abscess. The resulting scar from this procedure is seen in the region of the left trochanter. The method of delivery of this patient is not definitely decided on and a further study of cephalopelvic relationships will be made at term. It is likely, however, that cesarean section will be the procedure of choice.

ETIOLOGY

Naegele, in discussing the origin of the deformity, wrote "I confess that my original opinion about it, namely that the deformity originates neither from external force nor from disease but by a deviation in the original development, appears the most probable." However, somewhat later, in 1860, Thomas [3] stated that in certain cases inflammatory disease was responsible for the condition. Breus and Kolisko [4] in 1900 went even further and stated that the condition was always the result of inflammatory disease and the history would substantiate this fact. In 1929 Williams [5]

Fig. 3.—Appearance of pelvis in case 3.

stated that this may not apply to the individual case and in his case, as well as in that of Reinberger [6] in 1933, an embryonic defect was the only explanation of the deformity. Berry Hart [7] in 1917 summarized his views, stating that "The true Naegele and Robert pelves have not had a previous osteitis with resulting ankylosis in the region of the sacroiliac joints, followed by disturbed weight transmission. The pseudo-Naegele and pseudo-Robert pelves have had a previous osteitis in these regions and there the synostosis and atrophy are the result of this. The forms of the Naegele and Robert pelves are the result of polar losses of the size

Fig. 4.—Appearance of pelvis in case 4.

elements of the alae sacri and innominate bones, due to maturation of the sperm and germ cells. In these the loss of the alae sacri and innominate determinates has occurred, a great rarity, more often a unilateral loss (Naegele) than a bilateral one (Robert). The sacroiliac ankylosis is due to the fact that by such losses (bony elements and joint elements) the part remaining, imperfectly developing, becomes ankylosed. As this is a germ plasma change and multiplication of the reduced elements occurs, it may be transmitted."

In considering the cases here presented, it is interesting to note that in case 1 coexistent malformations were present and that they were definitely associated with the urogenital system, as in Reinberger's case, and that reported by the Rivières.[8] In case 2 there was nothing in the history or physical findings to suggest either associated malformation or previous disease. In case 3 the history of osteomyelitis during the first three years of life is not of great speculative interest, for the deformity occurred on the side opposite to the bone lesion. In case 4 the previous tuberculous lesion was on the same side as the deformity, but the structure of bone in the region of the sacroiliac region does not suggest any previous pathologic process.

DIAGNOSIS

Without the use of roentgen technics it is apparent that the diagnosis of Naegele pelvis in the living presents difficulties. In 2 of the foregoing cases in which childbirth had taken place it seems apparent that the true deformity was unsuspected. In case 1 the deformity was not discovered until roentgen technics were employed during a urologic investigation. An instance of the diagnostic difficulty by ordinary methods is seen in cases 2 and 3, in which numerous vaginal examinations were made by presumably competent observers. In all fairness to them, however, it should be pointed out that our knowledge of the frequent existence and the obstetric importance of midplane pelvic contractions has been emphasized only comparatively recently. The absence of obvious external deformity and change in gait also contribute to the obscurity of diagnosis. The

3. Thomas, A. E. S.: Arch. f. d. hölland. Beitr. z. Nat.- u. Heilk. Utrecht **2**: 270, 1860.
4. Breus, C., and Kolisko, A.: Die pathologischen Beckenformen, Leipzig, F. Deuticke, 1900.
5. Williams, J. W.: Am. J. Obst. & Gynec. **18**: 504, 1929.
6. Reinberger, J. R.: Am. J. Obst. & Gynec. **25**: 834, 1933.
7. Hart, D. B.: Edinburgh M. J. **18**: 4, 1917.
8. Rivière and Rivière: Bull. Soc. d'obst. et gynéc. de Paris **12**: 85, 1923.

transverse diameter of the outlet is always diminished, so that in examining vaginally there is a definite sense of constriction. In lateral vaginal palpation the pelvic wall on the affected side is found to be definitely displaced toward the midline. If vaginal palpation of the pelvic sidewalls was carefully made at least once during the antepartum period of every pregnant patient it is probable that the diagnosis would not be overlooked. At least a roentgen investigation would be suggested in such instances and the diagnosis would be settled thereby.

The importance of routine roentgen pelvimetry for every primigravid woman again becomes obvious, for if gross pelvic anomalies, such as Naegele pelvis, can be so easily overlooked, lesser but equally important pelvic changes will remain likewise undiscovered by the ordinary methods of investigation. Our experience with six years of routine roentgen pelvimetry for this group greatly strengthens this opinion.

SUMMARY

1. The observation of four Naegele type pelves within five years in a relatively limited amount of material demonstrates that this type of pelvic deformity is not of the extreme rarity formerly supposed.

2. Unless careful palpation of the pelvic side walls is done at vaginal examination, the condition apparently may be easily overlooked. Roentgen pelvimetry will always establish the diagnosis.

3. In some women with this type of deformity the birth of a living child through the natural passages is possible.

4. The importance of at least one roentgenometric pelvic survey for every primigravid woman is an obstetric ideal which will rapidly become realized when the advantages of the procedure are fully appreciated.

Clinical Notes, Suggestions and New Instruments

A CASE OF POLIOMYELITIS IN A NEWBORN INFANT

A. H. BIERMANN, M.D., GARDEN PLAIN, KAN., AND
E. A. PISZCZEK, M.D., CHICAGO

The age of susceptibility to poliomyelitis has been of great interest to students of the disease. The problem of fetal infection has been studied by Brahdy and Lenarsky,[1] by Kleinberg and Horwitz[2] and by Harmon and Hoyne,[3] who conclude that in pregnancy there is apparently a failure of fetal infection to occur during the course of poliomyelitis.

Cases occur infrequently in the infant under 6 months of age. This may be correlated with the findings of Shaughnessy, Harmon and Gordon[4] and of Aycock and Kramer,[5] who found that the infant usually shows antibodies if the mother has neutralizing antibodies in her blood. These appear to persist for several months after birth.

The incubation time of acute anterior poliomyelitis likewise has been a matter of much discussion among epidemiologists of the disease. The following case histories concern a mother and child who were victims of poliomyelitis during the 1943 season:

MOTHER'S HISTORY

Mrs. A. G., aged 26, entered the Wichita General Hospital on Oct. 23, 1943 at 1:25 a. m. with definite contractions of labor every five minutes, the pains lasting thirty to forty seconds. On admission the temperature of 98.2 F., pulse 78 and respirations 18 was recorded. Rectal examination at the time revealed 4 cm. dilatation of the cervix and outline well effaced. The history previous to these findings was negative except for a sore back for one day prior to admission to the hospital, which was believed due to the beginning of labor. At 2:30 a. m. contractions became stronger and 1½ grains (0.1 Gm.) of pentobarbital sodium was given. At 5:19 a. m. normal delivery of a 7 pound 3 ounce (3,260 Gm.) boy occurred.

The placenta was expressed intact and a second degree laceration repaired. The first rise in temperature was noted at 4 p. m., about eleven hours after delivery, at which time the temperature of 100.6 F., pulse 96, respirations 18 was recorded. On the first day after delivery the temperature was 100.6 F., pulse 84 and respirations 24. The patient complained of sore neck and back. The temperature rose to 101.6 F., pulse 100, respirations 24 at 10 a. m. The white blood cell count was 10,500. At noon the temperature was 102.2 F., pulse 116, respirations 24. At 2:30 p. m. a spinal puncture was done; the cell count was 65, polymorphonuclears 1, lymphocytes 64, positive Pandy test. The patient was removed to the poliomyelitis ward. At 4 o'clock the temperature rose to 103.6 F., pulse 116, respirations 30, and all other findings were inconsequential except that the patient complained of a sore neck and back which antedated to the day previous to admission to the hospital. At 10 p. m. the patient developed weakness in the right lower extremity. Kenny hot packs were started.

On the second day after delivery, October 25, the temperature fluctuated from 100.4 F. to 103 with dyspnea and gradually increasing cyanosis. At 8 a. m. there was definite paralysis of the right lower and upper extremities. At 4 p. m. the patient was very cyanotic and dyspneic; oxygen was started. At 8 o'clock complete paralysis of both lower extremities and right upper extremity was noted.

The third day after delivery, the 26th, the temperature fluctuated from 100.6 to 103 F. The patient was very nervous and cyanotic; complete hot packs to the body were applied. At 8 a. m. paralysis of both lower extremities and the right upper extremity was noted in addition to weakness of the left upper extremity. A white blood cell count at this time showed 12,200 cells. At noon, suction was started because of difficulty in swallowing (bulbar poliomyelitis signs) and by 3 p. m. the patient's color was much better and less mucus was present in the throat. At 8 o'clock an examination revealed complete paralysis of both upper and lower extremities. At 9:15 respirations became very shallow, accompanied by a weak pulse. The patient was placed in a respirator and oxygen continued by mask.

The fourth day after delivery, the 27th, the temperature fluctuated from 102.6 to 104.4 F. There was complete paralysis of both upper and lower extremities. The patient remained in the respirator, but the respirations were irregular and intermittent; during this time the patient was very listless, cold and at times irrational. A thorough examination at this time revealed no apparent clinical evidence of any complications of pregnancy. During the day suction was used frequently to remove mucus from the throat. The patient became gradually worse and died at 8:15 p. m. in the respirator. No autopsy was performed.

HISTORY OF THE CHILD

On the day of birth, Oct. 23, 1943, the child was admitted to the nursery in good condition. At 9 a. m. the child was seen by the mother for a ten minute period. No specific observation was made regarding the degree of contact between the mother and the child. The child was placed in bed beside

This case was discovered in the course of a survey of epidemics made for the National Foundation for Infantile Paralysis, Inc.

1. Brahdy, M. B., and Lenarsky, Maurice: Acute Epidemic Poliomyelitis Complicating Pregnancy, J. A. M. A. **101**: 195 (July 15) 1933.
2. Kleinberg, Samuel, and Horwitz, Thomas: The Obstetrical Experience of Women Paralyzed by Acute Anterior Poliomyelitis, Surg., Gynec. & Obst. **72**: 58 (Jan.) 1941.
3. Harmon, P. H., and Hoyne, Archibald: Poliomyelitis and Pregnancy, J. A. M. A. **123**: 185 (Sept. 25) 1943.
4. Shaughnessy, H. J.; Harmon, P. H., and Gordon, F. B.: The Neutralization of Poliomyelitis Virus by Human Serum, J. Prev. Med. **4**: 463, 1930.
5. Aycock, W. L., and Kramer, S. D.: Immunity to Poliomyelitis in Mothers and the Newborn as Shown by the Neutralization Test, J. Exper. Med. **52**: 457, 1930.

the mother. According to the history, this was the only contact between the mother and the child subsequent to the delivery.

The child progressed normally until the 11th day of life, November 2, when it became listless, cried seldom and at times was slightly cyanotic, showing some throat congestion. The temperature rose to 99.4 F. On the following day, the 3d, the condition of the child remained the same.

On November 4, the 13th day of life, there was noted definite weakness in both lower extremities. A spinal puncture revealed a cell count of 57, polymorphonuclear cells 12 and lymhocytes 45. The child was removed to the poliomyelitis ward.

On the 14th day of life there was inability to move the upper extremities in a normal manner.

On the 16th day of life improvement was noted in the right upper extremity, while the left upper extremity remained the same.

On November 8, the 17th day of life, both upper extremities and the left lower extremity showed improvement. The right lower extremity was completely paralyzed.

On November 20, the 29th day of life, general improvement was noted except for the right lower extremity, where there was only slight motion of the toes. There was pronounced bulging of the right side of the abdomen due to paralysis of the abdominal muscles.

On December 24, the 63d day of life, there was complete paralysis of the right lower extremity except for slight motion of the toes, with definite beginning muscular atrophy. The right abdominal muscle wall was bulging because of paralysis, requiring an abdominal binder; otherwise the child was gaining normally both mentally and physically.

This patient was a resident of the city of Wichita, in which county 164 cases of poliomyelitis occurred during the 1943 season. In the town district in which this patient resided there were 9 cases of poliomyelitis, the mother being the 7th case and the baby the 8th. The patient resided with her husband, aged 26, and 2 other children, aged 2 and 3. None of the other members of the family were ill at any time during the illness of the mother. The patient lived in an area of the town which was supplied by city water and a sewage disposal drainage system.

This infant began to show signs of poliomyelitis in its 11th day of life, which falls within the estimated period of incubation of six to twenty days as determined by Aycock and Luther.[6]

The problem arises "When was this infant exposed to the disease?" The following possibilities must be considered:

1. *Fetal Infection.*—This question has been studied thoroughly, and it is felt that failure of the disease to develop in the fetus of a mother with acute poliomyelitis is due to the low concentration of the virus in the blood stream or to its absence.[7]

2. *Birth Canal Exposure.*—Harmon,[8] Paul and Trask,[9] Howe and Bodian [10] and numerous other workers in the poliomyelitis field have shown the presence of poliomyelitis virus in the feces of a high percentage of poliomyelitis patients during the early stages of the disease. Despite modern asepsis, contact between the mother's feces and the newborn child is possible at the time of birth.

3. *Contact Exposure.*—Four hours after birth, in accordance with practice in this hospital, the infant had a ten minute contact with the mother while the latter was in the early stage of the disease. There is no record of the intimacy of this contact, but it is possible that either buccal, nasal or rectal secretion contact may have been possible between the mother and the child.

4. *Other Contact Exposure.*—It has been shown by Langmuir [11] and by Piszczek, Shaughnessy, Zichis and Levinson [12] and others that a high percentage of contacts to cases of poliomyelitis may become carriers of the disease, especially during a time when there is a high community prevalence of the disease. Other persons who came in contact with the newborn infant may have been carriers of poliomyelitis virus.

In the absence of laboratory evidence that contacts of the child other than the mother were excreting poliomyelitis virus, it is, of course, impossible to determine the source of the infant's infection; however, it appears to us that the available evidence would suggest that infection occurred at time of birth or shortly thereafter.

6. Aycock, W. L., and Luther, E. H.: The Incubation Period of Poliomyelitis, J. Prev. Med. **3**: 103, 1929.
7. The Question of Intrauterine Poliomyelitis, editorial, J. A. M. A. **123**: 210 (Sept. 25) 1943.
8. Harmon, P. H., in discussion of Toomey, John A.: Active and Passive Immunity and Port of Entry in Poliomyelitis, J. A. M. A. **109**: 402 (Aug. 7) 1937.
9. Paul, J. R., and Trask, J. D.: The Virus of Poliomyelitis in Stools and Sewage, J. A. M. A. **116**: 493 (Feb. 8) 1941.
10. Howe, H. A., and Bodian, David: Untreated Human Stools as a Source of Poliomyelitis Virus, J. Infect. Dis. **66**: 198-201 (May-June) 1940.
11. Langmuir, A. D.: Carriers and Abortive Cases in a Rural Poliomyelitis Outbreak, Am. J. Pub. Health **32**: 3 (March) 1942.
12. Piszczk, E. A.; Shaughnessy, H. J.; Zichis, Joseph, and Levinson, S. O.: Acute Anterior Poliomyelitis; Study of an Outbreak in West Suburban Cook County, J. A. M. A. **117**: 1926 (Dec. 6) 1941.

Council on Pharmacy and Chemistry

NEW AND NONOFFICIAL REMEDIES

THE FOLLOWING ADDITIONAL ARTICLES HAVE BEEN ACCEPTED AS CONFORMING TO THE RULES OF THE COUNCIL ON PHARMACY AND CHEMISTRY OF THE AMERICAN MEDICAL ASSOCIATION FOR ADMISSION TO NEW AND NONOFFICIAL REMEDIES. A COPY OF THE RULES ON WHICH THE COUNCIL BASES ITS ACTION WILL BE SENT ON APPLICATION.

AUSTIN E. SMITH, M.D., Secretary.

SOLUTION OF EPINEPHRINE HYDROCHLORIDE (See New and Nonofficial Remedies, 1943, p. 263).

The following products have been accepted:

BURROUGHS WELLCOME & CO., INC., NEW YORK

Solution of Epinephrine Hydrochloride 1: 1,000: 30 cc. bottle. Contains epinephrine hydrochloride 0.1 per cent, chlorobutanol 0.5 per cent, potassium metabisulfite 0.1 per cent and sodium chloride in isotonic solution.

Hypoloid Epinephrine Hydrochloride Injection: 1 cc. ampuls. Contains epinephrine hydrochloride 0.1 per cent, chlorobutanol 0.5 per cent, potassium metabisulfite 0.1 per cent and sodium chloride in isotonic solution.

THE WARREN-TEED PRODUCTS CO., COLUMBUS, OHIO

Sterilized Solution of Epinephrine Hydrochloride 1: 1,000: 30 cc. rubber stoppered vials. Contains epinephrine hydrochloride 0.1 per cent, sodium bisulfite 0.1 per cent and chlorobutanol 0.5 per cent in isotonic solution of sodium chloride.

SUSPENSION OF EPINEPHRINE IN OIL 1: 500 (See New and Nonofficial Remedies, 1943, p. 266).

The following product has been accepted:

ABBOTT LABORATORIES, NORTH CHICAGO, ILL.

Ampul Epinephrine in Oil 1: 500: 1 cc. A suspension of 2 mg. of epinephrine in 1 cc. of purified peanut oil.

EPHEDRINE SULFATE (See New and Nonofficial Remedies, 1943, p. 256).

The following dosage form has been accepted:

WILLIAM R. WARNER & CO., INC., NEW YORK

Ampul Solution Ephedrine Sulfate: 50 mg. in 1 cc.

SULFATHIAZOLE (See New and Nonofficial Remedies, 1943, p. 182).

The following product has been accepted:

WILLIAM R. WARNER & CO., INC., NEW YORK

Tablets Sulfathiazole: 0.5 Gm.

CONTRACEPTIVE DIAPHRAGMS (See THE JOURNAL, Dec. 18, 1943, p. 1046).

The following product has been accepted:

JULIUS SCHMID, INC., NEW YORK

Ramses Diaphragms. Gum rubber diaphragms covering a circular spring, the external diameter varying in gradations of 5 mm. from 50 to 100 mm.

U. S. Patent 2,024,539. U. S. Trademark 284,083.

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SATURDAY, JANUARY 29, 1944

DETECTING THE EXACT TIME OF OVULATION BY OVULATION POTENTIALS

Statistical studies that correlate menstruation with coitus and fertility apparently confirm the conclusion drawn from endocrine and anatomic studies that ovulation occurs in a strictly limited time in relation to subsequent menstruation. The duration of ovulation time thus indicated, according to Rock and his associates,[1] is still a matter of four or five days, during which the ovum is susceptible to fertilization for only a few hours. The uncertainty is further increased by the fact that the date of subsequent menstruation from which one may estimate ovulation time is predictable only within a wide range of several days or weeks. The importance of being able to determine the exact time of ovulation, both for conception and for contraception, is evident.

Recent studies of the problem of the so-called ovulation potentials seemed to promise a solution. In 1935 Burr, Hill and Allen detected in rabbits by means of a vacuum tube potentiometer a definite difference of potential between a suprapubic and a vaginal electrode when each ovulation occurred. Similar observations were made by Reboul, Friedgood and Davis in rabbits, by Rogers at the time of estrus in the rat and by Altmann in the hen at the time of ovulation. Burr and his associates described the same phenomenon in the human female, using an abdominovaginal circuit, and Rock and his associates obtained the same results in 10 women during the theoretical ovulation period.

Now a more careful analysis by Snodgrass, Rock and Menkin[2] of their results convinces them that there existed a number of discrepancies which made the value of this "abdominovaginal sign" of the time of ovulation questionable. The potential change in some cases was observed even before ovulation had occurred; there were wide variations in the magnitude, duration and polarity of the electrical changes; erratic results were obtained when surface electrodes were employed simultaneously with an abdominovaginal circuit. Finally it was difficult to explain how the potential changes could increase in magnitude as the electrodes were moved farther away from the ovary. The observation of Rogers of a similar change in difference of potential on the last day of estrogen induced estrus in the spayed rat further increased their skepticism as to the relation between the so-called abdominovaginal sign and ovulation. In their more recent experiments Snodgrass, Rock and Menkin attempted to determine whether one could locate a point on the surface which would be stable in potential and could thus serve as the site of a reference electrode. From seventy-five to a hundred determinations of potential were made on each subject. When the potentials were sufficiently stable it was possible to construct a clearly defined series of isopotential contours. It was found that there was no demonstrable effect of sex on isopotential contours. A focus of potential could be found on the thighs in the region where the femoral artery and vein are most superficial. Potential foci could also be observed at almost any point on the abdomen, and they did not seem to have any demonstrable connection with the underlying organs. Sthenic types of individuals usually had unstable potentials. It appeared that the reason for the instability was due entirely to emotional uneasiness of the subject. Direct electrical measurements were made on human ovaries in situ on 3 women in the course of routine laparotomy. Each operation was performed within the patient's estimated ovulation time. No potentials greater than ± 1 millivolt were found between any two organs in the abdominal cavity. Several follicular surfaces, as well as the intervening ovarian cortex, were tested, but no significant electrical potential was detected. The highest potential recorded, slightly less than $+2$ millivolts, was obtained from a point on the rectus abdominis muscle. The authors further examined the so-called finger to finger potentials. Experiments on 30 nurses and 23 female employees of the Free Hospital for Women revealed that there is a relationship of polarity to "handedness." The authors were at a loss to explain the constancy of opposite polarity in finger potentials related to "handedness" and the theoretical influence of the ovaries.

In summarizing their experiments, Snodgrass and his associates state that the difficulty of locating on the skin surface a stable reference electrode is in itself a rational explanation for the inconsistent results previously obtained in connection with the so-called abdominovaginal sign of ovulation. The influence of temperature on the potential difference curves demonstrated a definite relation between thermal and electrical changes and indicated that the mechanism by which temperature influences potentials is concerned, at least in part, with changes in p_H. Since skin temperature

1. Rock, John; Reboul, Jean, and Snodgrass, James M.: Electrical Changes Associated with Human Ovulation, Am. J. Obst. & Gynec. **36**: 733 (Nov.) 1938.

2. Snodgrass, James M.; Rock, John, and Menkin, Miriam F.: The Validity of "Ovulation Potentials," Am. J. Physiol. **140**:394 (Dec. 1) 1943.

fluctuations are known to be induced by vasomotor and endocrine changes, it seems probable that the so-called ovulation potentials previously reported, whether elicited between a vaginal and a suprapubic electrode, between two surface areas—abdominal or flank—or between fingers, are measurements only of local changes in peripheral cutaneous blood flow due to alterations in capillary tone. These authors conclude that the bioelectrical phenomena previously reported as related to ovulation are due primarily to cutaneous vascular conditions and possibly only remotely and to a slight degree to ovarian influence. Local changes in blood flow affect the focal temperature, which in turn reacts on the focal p_H, the fluctuations of which directly determine potential variations. Because the endocrine factor is only one of several affecting vascular phenomena, they believe that potential changes recorded thus far cannot be attributed solely to an ovarian activity and that therefore the electrical methods heretofore proposed are unsuitable for the detection of ovulation.

AUSTRALIAN GOVERNMENT ACCEPTS MEDICAL ADVICE

The leaders of the Australian government now admit that the administration was too hasty in its attempt to perpetrate a wholesale transformation of health services and to abolish private practice.[1] The original proposal called for a whole time salaried medical service with district organizations and an elaborate system of promotions and integration of all health services. Extensive discussion modified many phases of this plan but left it still cumbersome and bureaucratic. The interim report now states that "The committee is impressed by the existing widespread opposition to the proclamation of this act and concludes from it that the scheme there suggested is unsuitable as a permanent basis for social benefits or health services for the people of Australia."

The committee, after considering the published reports of certain of these meetings and the evidence given before it by witnesses, believes:

1. That a substantial majority of medical practitioners object to a general salaried medical service on lines laid down by the National Health and Medical Research Council or any other lines put forward up to date.

2. That, on the other hand, there is general agreement that a salaried medical service may be justified in remote areas.

3. That there is practical unanimity of opposition from all private medical witnesses to any proposal for control of any general health service by any government department; and support for control by an independent body or commission, including a majority of medical men, if such a general health service should be introduced.

4. That there are indications that a more favorable attitude toward a salaried medical service may exist among medical officers of the fighting services; but it is not possible accurately to assess the situation without consulting every member individually.

The report considers at considerable length the desirability of expansion of preventive health measures and provisions for dealing with tuberculosis and mental and venereal disease. Regarding general medical service, it is considered "that a general medical service should be instituted, as the best and most equitable means of providing medical care for the community as a whole; and that this should be financed from a central fund specifically raised for the purpose by a tax on income, having regard to the capacity of the individual to pay."

As to the methods of paying physicians, the merits and demerits of capitation, panel and fee for service plans are considered, but a definite recommendation is not made.

On methods of control and administration the report says:

> While, therefore, there is pronounced opposition to the scheme outlined by the National Health and Medical Research Council which visualizes a system of governmental control of all health services, there is considerably less opposition to such a service if control is vested largely in the medical profession through an independent body with statutory authority and removed from political control. Indeed, upon this aspect of administration there is almost complete unanimity. Such freedom from political control is essential for the success of any scheme. Undoubtedly, a large section of opinion regards a full time salaried medical service as a revolutionary proposal which might seriously affect the medical profession and its services and, in fact, the existing social order. For this also they oppose it.

Still maintaining "that the ultimate solution will probably be found in a full time salaried medical service," it concludes that:

> Having regard to all the circumstances and to the need for early and substantial reorganization of and improvement in health services generally as indicated therein, we consider that, subject to further discussion, . . . such services should now be planned, for introduction as and when the war situation permits, as follows:
>
> 1. *For Remote Areas* (i. e., areas which now have difficulty in maintaining one general practitioner or where difficulty is experienced in securing adequate medical services) *a Voluntary Full Time Salaried Medical Service* under a limited term appointment; with improved hospital and transport services, including extended ambulance and flying doctor services and facilities for consultant services; such services to be established and extended as necessary.
>
> 2. *For All Other Areas* (i. e., cities and country towns) *a Part Time Salaried Medical Service,* under a system of voluntary participation by general practitioners who would retain their private practices and would nominate the number of half-day sessions they would be willing to devote to a general medical service on a part time salaried basis. Such service should be provided at outpatient and consulting clinics located in the centers of population in suburban areas and country towns. Clinics would be equipped with all modern diagnostic aids and treatment facilities and would be supervised by a salaried Medical Liaison Officer responsible to the Central Health Administration. General control of the clinics would be vested in the medical personnel of each clinic.

A further suggestion proposes that the plan should be administered under a commission, a majority of whom should be nominated by the medical profession. Thus the Australian government continues to grope

1. The Parliamentary Joint Committee on Social Security: Sixth Interim Report on a Comprehensive Health Scheme, M. J. Australia 2: 41 (July 17) 1943.

toward some revolutionary all-embracing scheme for medical practice, failing to realize that a wide diversity of social, economic and scientific facilities must inevitably demand a diverse approach in the delivery of medical service.

Current Comment

SMALLPOX IN RELATION TO VACCINATION LAWS

Vaccination prevents smallpox; every doctor knows this! Repetition is necessary because of public apathy and antivaccination movements. Public apathy results from the fact that smallpox has not in recent years appeared in serious form or in widespread or locally intensive or explosive outbreaks. The United States Public Health Service reports [1] a sharp decrease in smallpox in the United States since 1931, especially during the years 1940 to 1942 inclusive. The service reports "There still remains, however, a significant inverse correlation between the incidence of the disease and the rigorousness of the provisions of law or regulations regarding vaccination. This correlation is especially marked when the incidence rates are compared by states grouped according to the positiveness of their vaccination requirements." The accompanying table shows incidence rates for smallpox by groups of states according to their vaccination requirements. Smallpox is obviously lowest in those jurisdictions where some type of universal routine vaccination requirement is in effect. Such studies as this are effective refutation of claims made by groups opposed to vaccination that vaccination is available for those who want it and no one else should be compelled to have it. In those areas where vaccination requirements are lax the people will continue to pay the price for needless prevalence of a completely preventable disease.

Average Smallpox Case Rates, 1938-1941

Group and States included	Annual Case Rate per 100,000 Population
1. States (13, including the District of Columbia) requiring vaccination of pupils as a prerequisite to school attendance, regardless of the presence or absence of smallpox (Arkansas, Kentucky, Maryland, Massachusetts, New Hampshire, New Mexico, New York, Pennsylvania, Rhode Island, South Carolina, Virginia, West Virginia, District of Columbia)	0.8
2. States (6) in which vaccination of pupils may be required at all times (Connecticut, Georgia, Maine, New Jersey, Ohio, Oregon)	3.0
3. States (10) having various permissive provisions regarding vaccination (Alabama, Colorado, Connecticut, Georgia, Kansas, Michigan, Mississippi, North Carolina, Tennessee, Wyoming)	3.6
4. States (12) having varying provisions which direct or authorize the exclusion of unvaccinated persons from school only when smallpox is present or threatened (Arizona, Iowa, Kansas, Louisiana, Minnesota, Montana, Nebraska, New York, North Carolina, Oregon, Texas, Wisconsin)	6.3
5. States (9) which have no important laws or regulations promoting or efficacious in achieving the application of vaccination of the population (Delaware, Florida, Idaho, Illinois, Indiana, Missouri, Nevada, Oklahoma, Vermont)	11.1
6. States (7) having various prohibitive provisions regarding the requirement of smallpox vaccination (Arizona, California, Minnesota, North Dakota, South Dakota, Utah, Washington)	13.2

1. Hampton, Brock C.: Smallpox in Relation to State Vaccination Laws and Regulations, Pub. Health Rep. **58**: 1771 (Dec. 3) 1943.

WARNING ON LABEL STATEMENTS

All too frequently accidents occur because drug labels are not read carefully prior to the administration. Accidents often follow the use of drugs when the labels do not offer sufficient warning statements. The possibility of severe reactions and even death is increased when potent agents intended only for subcutaneous or intramuscular use are administered intravenously; once such compounds are injected into the body, they are frequently beyond effective control. The Council on Pharmacy and Chemistry scans carefully the label statements of products submitted for inclusion in New and Nonofficial Remedies and demands the presence of adequate warnings if indicated. Frequently the Council considers the labels and other informative material of nonsubmitted products because of reports of accidents following their use. Twice in the past year one firm has been informed by the Council of fatalities following incorrect usage of two of its products which were not Council accepted. One fatality concerned a gold salt intended only for intramuscular use; the drug was administered intravenously. Specimens were procured on the open market; neither the label on the package nor the individual container contained a warning about intravenous use; nor was there an accompanying package insert to provide such information. The other product is a stimulant suitable for subcutaneous use. Purchased specimens provided 1 cc. ampules which carried in small black letters the statement "Do not use intravenously." The label on the package container did not carry a cautionary statement, and there was no accompanying package insert. While in each instance the firm adopted an immediate cooperative attitude, accidents because of inadequate labeling have occurred; the damage cannot be undone. The manufacturers seem to be increasingly conscious of responsibilities to the medical profession and the public. Nevertheless every one should aid in making certain that labels bear all necessary warnings. Such action will save lives!

SOCIAL HISTORY AND SELECTION FOR MILITARY SERVICE

An elaborate technic to aid in the preinduction selection of registrants for military service has been recently described by Frankel.[1] The program that is employed in New Jersey utilizes information already available from state welfare and health organizations, community agencies and other sources which may be concerned with the medical and social backgrounds of selective service registrants. The most important information that can be gained in this way refers to the mental institutional background of the registrant and of members of his family and to previous adult correctional institutional or tuberculosis sanatorium experience. Although the final decision for acceptance or rejection for military service of a registrant rests with the induction station, the degree of correlation between registrants with unfavorable institutional backgrounds and rejections by the induction stations has been high.

1. Frankel, Emil: Social Histories as Aids in the Selection of Members of the Armed Forces, State of New Jersey Department Institutions and Agencies, Trenton, December 1943.

MEDICINE AND THE WAR

In this section of The Journal each week will appear official notices by the Committee on War Participation of the American Medical Association, announcements by the Surgeons General of the Army, Navy and Public Health Service, and other governmental agencies dealing with medicine and the war, and such other information and announcements as will be useful to the medical profession.

ARMY

ARMY TO TURN OVER CERTAIN CAMPS TO VETERANS' BUREAU FOR HOSPITALS

The War Department announced recently that certain army camps which have been vacated by troops sent overseas will be turned over to the Veterans' Bureau for use as Veterans' hospitals. This decision was made at a meeting called by Lieut. Gen. Brehon Somervell, commanding general, Army Service Forces, in his office and attended by Brig. Gen. Frank T. Hines, chief of the Veterans' Administration, and Major Gen. Norman T. Kirk, Surgeon General of the Army. In announcing this transfer, General Somervell said that "the War Department is determined to see that every discharged, wounded or sick veteran gets the best possible care and that this care is uninterrupted. Anything less is unthinkable. Not a single veteran of the present war should be discharged until he is entirely recovered or until the Veterans' Administration has a bed for him. The number of beds in army hospitals is of necessity limited. Adequate preparation must be made to receive wounded men from overseas for immediate treatment. General Hines desires to assume responsibility for each veteran at the earliest possible minute. We are in complete agreement on the obligation of the government to the sick or wounded soldier. However, the Veterans' Administration has been hampered by lack of facilities and of trained staffs. Therefore we offered to turn over as many camps as General Hines needs to assure uninterrupted care and comfort for our men. He accepted our offer and we now are working out details together."

General Somervell announced at the same time that, in order to prevent doctors assigned to Veterans' hospitals from resigning to join the army, all such doctors who can pass physical examinations will be commissioned in the Army Medical Corps and reassigned to the Veterans' hospitals to assist General Hines in meeting the existing acute situation in regard to all types of personnel. Which military establishments will be transferred has not yet been decided, but the War Department and Veterans' Bureau officials hope to make their decision shortly and begin preparations to receive patients as rapidly as possible.

FOUR ARMY NURSES TO RETIRE

Capt. Frances M. Steele will retire from the Army Nurse Corps on March 31 after thirty-five years of service. After her training at the Maryland General Hospital in Baltimore she served one term in the Army Nurse Corps from 1907 to 1910, then resigned and reapplied one year later, this time for thirty-two years of service. During the first world war she was with an orthopedic unit at Ellis Island, New York, and also on duty with Major Stimson in the Surgeon General's Office, Washington, D. C. She has also seen duty in Hawaii, the Philippines, Puerto Rico and the Virgin Islands. Captain Steele was formerly principal chief nurse at the William Beaumont General Hospital, El Paso, Texas.

Lieut. Col. Anna A. Montgomery, after twenty-five years of service in the Army Nurse Corps, will retire on March 31. Colonel Montgomery, in addition to service in the United States, spent four years in the Philippines and accompanied the American Relief Expedition when the United States went to the aid of Japan after the earthquake there in 1923. Colonel Montgomery was formerly in charge of the nursing service of the Seventh Service Command, Omaha.

First Lieut. Alice I. Akeley will retire from active duty in the Army Nurse Corps on March 31, after twenty-five years of service. In addition to service in the United States, she served in Hawaii and the Philippines and was sent to Japan in 1923 from Manila with the American Relief Expedition following the earthquake of that year. During the first world war she served with Base Hospital No. 59 at Rimaucourt, France. Lieutenant Akeley was formerly in charge of nurses at the Darnall General Hospital, Danville, Ky.

First Lieut. Annie L. Baird, who has served twenty-five years in the Army Nurse Corps, will retire on March 31 because of ill health. Lieutenant Baird joined the Army Nurse Corps in 1918 at Fort Myer, Virginia, served in the Army of Occupation, Coblenz, Germany, in 1920, at Schofield Barracks, Hawaii, 1925-1928, and at Sternberg General Hospital, Manila, 1932 to 1936, in addition to various posts in the United States.

CAPT. EDGAR O. HUGHES AWARDED SOLDIER'S MEDAL

Capt. Edgar O. Hughes, formerly of Washington, Ill., was awarded the Soldier's Medal for "heroism on July 5, 1943. Fifteen freight cars loaded with gasoline, bombs and other military equipment were on a railroad siding near a station. Gasoline which was being transferred to two trailers suddenly ignited, causing a tremendous explosion. Rushing to the fire, Captain Hughes and others helped combat the flames in an effort to check the possibilities of further explosions and damage. He assisted in uncoupling the eighth and ninth cars and, using the cab of the gasoline trailer as a locomotive, Captain Hughes and his companions brought eight freight cars to safety. While they were attempting to repeat the process with respect to other cars near the burning gasoline some of the bombs exploded, the concussion knocking the men to the ground. Although suffering shock and abrasions, they continued their attempts to halt the spreading flames, which had now reached the nearby dry fields. Soon after, they were removed from the scene by superior command and given medical treatment. By his unfailing devotion to duty and outstanding courage, Captain Hughes was greatly responsible for saving from complete destruction vast quantities of military equipment. His clear thinking and unusual initiative have reflected great credit upon himself and have upheld the highest traditions of the military service of the United States."

Dr. Hughes graduated from the University of Illinois College of Medicine in 1941 and entered the service Feb. 26, 1942.

REGIONAL FRACTURE-ORTHOPEDIC CONFERENCE

A regional fracture-orthopedic conference was held at the Station Hospital, Mitchel Field, New York, November 22-23, with 270 army, navy and civilian personnel in attendance. The program was set up by the Army Air Forces for the Northeastern Area. Major Stuart Z. Hawkes, chief of surgical service, Mitchel Field, was chairman. An address of welcome was delivered by Lieut. Col. Chester S. Fresh, post surgeon, after which many papers were read on numerous kinds of fractures and other related subjects. A discussion followed each of the papers presented.

PROMOTIONS IN ARMY NURSE CORPS

The War Department announced recently promotions in both the Reserve and Regular Army Nurse Corps. The three army reserve nurses to be promoted to the rank of major, in accordance with the authorization for increased grades in the nurse corps, were Major Kathleen H. Atto, Major Edna B. Groppe and Major Mary C. Walker. Major Atto, who has been consultant to Col. Florence A. Blanchfield, superintendent, Army Nurse Corps, on psychiatric nursing and public informant on nurse procurement, has been transferred to the Second Command, with headquarters at Governors Island, N. Y., to head nursing service at that headquarters. Major Groppe is on duty in the Office of the Superintendent, Army Nurse Corps, where she is in charge of nurses' assignments overseas. Major Walker was named recently to take charge of the Army's Senior Cadet Nurse Program. Other promotions of reserve nurses were 1st Lieut. Gertrude Roberts, La Junta, Colo., to the relative rank of captain; 2d Lieut. Helen A. Smathers Agronsky, Big Run, Pa., and 2d Lieut. Emily E. McMullan Norton, New York, both to the relative rank of first lieutenant.

In the Regular Army Nurse Corps Major Mary G. Phillips, executive officer to Colonel Blanchfield, Capt. L. Gertrude Thompson, principal chief nurse at Walter Reed General Hospital, Washington, D. C., and chief nurse of the Military District of Washington, were both promoted to the relative rank of lieutenant colonel. Capt. Edith A. Aynes, now representing the Army Nurse Corps, Office of Technical Information, Surgeon General's Office, was promoted to the relative rank of major.

MEDAL OF HONOR AWARDED TO MEDICAL AID MAN

Pfc. Frank J. Petrarca, a medical aid man in an infantry company of the 145th Infantry, 37th Division, was awarded the Medal of Honor. The citation accompanying the award reads "Even on the threshold of death, he continued to display valor and contempt for the foe; raising himself to his knees, this intrepid soldier shouted defiance at the enemy, made a last attempt to reach his wounded comrade and fell in glorious death." The Medal of Honor was presented to his mother by Major Gen. Charles L. Scott, commanding general of the Armored Command, December 23. Private Petrarca's home was in Cleveland.

NAVY

CLASS OF NAVAL MEDICAL OFFICERS

The following medical officers completed the basic course of instruction at the Naval Medical School, National Naval Medical Center, Bethesda, Md., December 27. Included are the dates and places at which these officers served their internships. All these officers at the time of the graduation were lieutenants (jg) except Dr. John D. Langston, whose rank was that of lieutenant:

Agamy, Glen Nordwin Peter, MC-V(G), USNR, U. S. Marine Hospital, New Orleans, 1942-43
Alden, Manning Wallace, (MC), USN, Garfield Memorial Hospital, Washington, D. C., 1942-43
Bloom, George Robert, MC-V(G), USNR, U. S. Marine Hospital, New Orleans, 1942-43.
Buchanan, Francis Randall, MC-V(G), USNR, St. Lukes Hospital, Duluth, Minn., 1942-43
Connell, James Vincent, MC-V(G), USNR, Cooper Hospital, Camden, N. J., 1942-43.
Crocker, Harvey James, MC-V(G), USNR, Beverly Hospital, Beverly, Mass., 1942-43
Cronk, Gary Arnold, MC-V(S), USNR, Syracuse University Hospital, Syracuse, N. Y., 1939-41
Davol, Rector Thomson, MC-V(G), USNR, Bellevue Hospital, New York, 1941-42
Dupler, Donald Alphoeus (MC), USN, The Graduate Hospital, University of Pennsylvania, Philadelphia, 1940-42
Gardiner, Frederick Schneerer (MC), USN, St. Lukes Hospital, Cleveland, 1939-40
Gaspar, Max Raymond (MC), USN, Los Angeles County General Hospital, Los Angeles, 1941-42
Harer, George Albert, MC-V(G), USNR, Meyer Memorial Hospital, Buffalo, 1940-41
Hinkle, Lawrence Earl, Jr., MC-V(G), USNR, Peter B. Brigham Hospital, Boston, 1942-43
Hyatt, Robert Whittaker, MC-V(G), USNR, Episcopal Hospital, Philadelphia, 1942-43
Lanier, Joe Ellis, MC-V(G), USNR, St. Vincent's Hospital, Jacksonville, Fla., 1941-42
Langston, John Donald, Lieut. (MC), USN, Jefferson Hospital, Philadelphia, 1940-42
Machles, Fannie, MC-V(G) (F), USNR, Jersey City Hospital, Jersey City, N. J., 1942-43
Morris, Stanley Frederick, MC-V(G), USNR, Greenpoint Hospital, Brooklyn, 1938-40
Mountain, David Charles, MC-V(G), USNR, Hartford Municipal Hospital, Hartford, Conn., 1941-42
Romney, Seymour L., MC-V(G), USNR, Beth Israel Hospital, Boston, 1942-43
Saraniero, Gioconda Rita, MC-V(S) (F), USNR, St. James Hospital, Newark, N. J., 1935-36
Slack, Louise Wetherill, MC-V(G) (F), USNR, Lancaster General Hospital, Lancaster, Pa., 1936-37
Smith, John Puntenney, MC-V(G), USNR, Strong Memorial Hospital, Rochester, N. Y., 1942-43
Smith, Joseph Huter II, MC-V(S), USNR, Hahnemann Hospital, Philadelphia, 1937-38
Snider, John Schurr, MC-V(S), USNR, Staten Island Hospital, Staten Island, N. Y., 1939-41
Stephenson, Robert Hilton, MC-V(S), USNR, Grady Hospital, Atlanta, Ga., 1939-40
Train, John Kirk, Jr., MC-V(S), USNR, Bellevue Hospital, New York, 1938-40
Vose, Francis Prue, MC-V(G), USNR, Rhode Island Hospital, Providence, R. I., 1942-43

NAVAL OFFICER RECEIVES LEGION OF MERIT

The Legion of Merit has been awarded by the commander of the United States Naval Forces, Northwest African Waters, to Lieut. George M. Caldwell, formerly of White Sulphur Springs, W. Va., for exceptionally meritorious conduct as medical officer on the U. S. S. *Shubrick* during the Sicilian campaign. The citation accompanying the award read as follows:

"When the U. S. S. *Shubrick* was struck by an enemy bomb on Aug. 4, 1943 in the harbor of Palermo, Sicily, the ship was badly damaged with loss of all light and ventilation. Lieutenant Caldwell, with great coolness and professional skill, administered medical attention to the many critically wounded. His foresight and competence in preparation for handling casualties under battle conditions minimized the loss of life in this engagement.

"The extraordinary ability and outstanding devotion to duty displayed by Lieutenant Caldwell reflected great credit upon himself and the naval service."

Dr. Caldwell graduated from the University of Virginia Department of Medicine, Charlottesville, in 1935 and entered the service Sept. 21, 1942.

FIRST NAVAL MOBILE HOSPITAL DECOMMISSIONED

The first naval mobile hospital was decommissioned at Bermuda Oct. 5, 1943, exactly three years after its official birth. Naval Mobile Hospital No. 1 sailed for Guantanamo Bay, Cuba, Oct. 25, 1940 and served at that station until July 20, 1941, when the unit, including its equipment and some prefabricated buildings, sailed for Bermuda. The hospital was open for business at the new base on Sept. 1, 1941. With the completion of a permanent dispensary at the Naval Air Station, Bermuda, the need for the mobile unit no longer existed at Bermuda, and it was decommissioned on Oct. 5, 1942. Its 19 officers and 225 enlisted men were assigned to other duties, some to mobile hospitals in other theaters.

OPERATION DONE BY FLASHLIGHT SAVES MARINE

Lieut. Leo J. Koscinski, formerly of Chicago, was credited in a recent dispatch from Bougainville with helping to save the life of a young marine by an operation performed during the recent landing on Cape Torokina. Shrapnel had pierced the stomach, and death from internal hemorrhage seemed imminent. Because of an air raid alarm at that time, it was necessary to perform the operation, which saved the patient's life, by the light of four flashlights and under a suspended blanket. Dr. Koscinski graduated from Northwestern University School of Medicine, Chicago, in 1941 and entered the service April 14, 1942.

OPEN FIRST HOSPITAL CORPS SCHOOL FOR WOMEN

The first Hospital Corps School for enlisted members of the Women's Reserve, U. S. Naval Reserve, located at the National Naval Medical Center, Bethesda, Md., was opened on January 12. The new school has a capacity of 480 and will give a four weeks course. Since the first women were selected for corpsman duty on Jan. 22, 1943 the training has been given in small groups at seventeen different naval hospitals. With the opening of the new school, training enlisted women for Hospital Corps duty will be concentrated there. The hospitals will give three weeks of ward duty experience to graduates of the Corpsmen's School. The curriculum of the new school, which is substantially the same as for men of the Hospital Corps, will include courses in anatomy and physiology, first aid and minor surgery, hygiene and sanitation, nursing, metrology and pharmacology. When these courses plus the three weeks active ward duty are completed, graduates may be rated from hospital apprentice, second class, to pharmacist's mate, second class, depending on their abilities and experience. Two hundred and forty enlisted women are scheduled to enter the Hospital Corps School every two weeks, and an additional 60, who are already qualified by civilian experience as technicians, laboratory assistants or nurse's aides, will be sent directly from "boot camp" at the U. S. Naval Training School, the Bronx, N. Y., to the St. Albans, N. Y., or San Diego Naval Hospitals for special courses. On completion of this training they may be rated from hospital apprentice, first class, to pharmacist's mate, second class. Women Reservists are also eligible for all advance Hospital Corps training courses offered to men except those in deep sea diving, certain phases of aviation medicine and the medical field service. These courses include clerical procedure, dental technology, operating room technic, fever therapy, physical therapy, low pressure chamber work, roentgenology, neuropsychiatry, epidemiology and sanitation, malariology and pharmacy and chemistry.

The staff of the new Corpsmen's School will be: Capt. John Harper, commanding officer; Comdr. Warren G. Wieand (MC), U.S.N., executive officer; Lieut. (jg) Ruth Roberta Mason, U.S.N.R., disbursing officer; Lieut. (jg) Clarence W. Ferguson, Hospital Corps, U.S.N., personnel officer; Lieut. (jg) Clara Alice Stolp, Nurse Corps, U.S.N., chief nursing instructor; chief pharmacist Eugene P. Campbell, U.S.N, chief instructor.

LIEUT. COMDR. JAMES F. STANDARD AWARDED SILVER STAR MEDAL

The Silver Star Medal was recently awarded to Lieut. Comdr. James F. Standard "for conspicuous gallantry and intrepidity in action against the enemy while serving with a Marine aircraft wing on Guadalcanal Island on the night of Oct. 13-14, 1942. During the vicious night attack against our positions on the island by enemy naval units, which commenced an unprecedented shelling together with sporadic bombings, Lieutenant Standard, with complete disregard for his own safety, proceeded to the sick bay and then led an ambulance through the shelled area to assist in giving aid to the wounded. His courageous conduct was in keeping with the highest traditions of the United States Naval Service." Dr. Standard graduated from Washington University School of Medicine, St. Louis, in 1936 and entered the service on Feb. 10, 1941. He is now serving on active duty for the second time somewhere in the South Pacific.

MISCELLANEOUS

BOOKS FOR HOSPITALIZED SOLDIERS

Margaret Willis
Hospital Librarian
JEFFERSON BARRACKS, MO.

Books, more than anything else, are today influencing the thoughts and feelings of soldiers confined to army hospitals. Frankly, as librarian at Jefferson Barracks, basic training center No. 1 of the AAF Training Command, I am amazed at the serious interest of many soldiers in the more profound books. The soldiers do not hesitate to express their delight when they find such books in the library and their chagrin if they do not. Requests come in daily for the philosophy of Santayana, Spinoza, Plato, Aristotle, Nietzsche, Schopenhauer, John Dewey, Thorstein Veblen, Karl Marx and Lin Yutang as well as of Jefferson, Lincoln and Walt Whitman. These soldiers seem deeply concerned with what outstanding writers have thought about life, its meaning and spiritual possibilities.

"Bed patients" read constantly because they can do nothing else. To these boys a book to their liking is a vacation from monotonous surroundings. While reading the special book they have chosen they forget themselves—a basic requisite in the building of morale. As an influence of modern thought, good recreational reading should not be dismissed too lightly. Not all important ideas are stated directly in so many words. "For Whom the Bell Tolls" or "So Little Time" say a great deal about our times indirectly. Hospitalized soldiers discover in "Grapes of Wrath," "Native Son," "Brothers Under the Skin" and "A Tree Grows in Brooklyn" the problems of the other fellow. As a result, through their better understanding of the needs of all classes and races, soldiers will be more inclined to contribute to the coming peace rather than to hinder it.

THIRTY-EIGHT A. M. A. EMPLOYEES NOW IN THE ARMED FORCES

Up to the present time thirty-eight employees of the American Medical Association have entered the armed forces of the United States, ten of whom are now overseas. One of them, Master Sergt. John A. Kovacs, member of the staff of the Council on Pharmacy and Chemistry before going into the army, has been awarded the Legion of Merit (THE JOURNAL, Dec. 18, 1943, p. 1054). The assistant editor of THE JOURNAL, Lieut. Col. J. F. Hammond, a retired regular army officer who has been with the Association since 1922, was called to active duty on Nov. 9, 1942 as a member of a permanent Court Martial in the Sixth Service Command, serving in that capacity until about Sept. 1, 1943, when he was appointed editor of the *Bulletin of the U. S. Army Medical Department*, Washington, D. C. A list of the other employees, together with the last known address, is given under the respective branch with which they are serving:

ARMY

Pvt. Harry Amici, 2d Med. Sup. Depot, 1st Advance Section, U. S. Army, APO 302, % Postmaster, New York.

Pvt. Victor Amici, Station Hospital Med. Det., Camp Chaffee, Ark.

S/Sgt. Marshall B. Barry, 46th Depot Repair Squadron, APO 635, % Postmaster, New York.

Pfc. Arthur Bierwirth, 448 Bomb. Gp. Squadron 712, Wendover Field, Utah.

Capt. William B. Bradley, A. C., 24th Altitude Training Unit, Selman Field, Monroe, La.

Pvt. James A. Dale, Med. Det. 322 Inf., APO 81, Camp San Luis Obispo, Calif.

Pvt. Gus Christodolos, 744 W. North Avenue, Chicago.

Cpl. Fred Grabenhofer, 490 Engineers Equipment Company, APO 4580, % Postmaster, San Francisco.

Pvt. George E. Hall Jr., Barracks 2427, 2d Platoon, Troop N, 2d Regt., C. R. T. C., Fort Riley, Kansas.

Lieut. Col. J. F. Hammond, Surgeon General's Office, Washington, D. C.

Pfc. Robert Jaeger, Wagon Co. 1624-S. U., Camp Ellis, Ill.

Pvt. Thomas Judge, Co. D, 728 M. P. Bn., Camp River Rouge Park, Detroit.

Capt. Harold D. Kautz, M. C., 67th Station Hospital, APO 606 % Postmaster, Miami, Fla.

Pvt. Frank Konieczny, Service Btry, 83 F. A. Bn., Fort Sill, Oklahoma.

M/Sgt. John A. Kovacs, Medical Section, N. A. T. O., U. S. Army, APO 534, % Postmaster, New York.

Pfc. Fred. H. Lech, APO 4937, % Postmaster, New York.

Pfc. Arthur McCurdy, 476 Flex. Gun. Train. Squadron, Harlingen Army Air Field, Harlingen, Texas.

Pfc. Charles Moose, Gunnery Sqd. #6 Class 44-5 Y. A. A. F., Yuma, Arizona.

Pfc. George Olsen, 69th Signal Bn., Co. C, Camp McKin, Grenada, Miss.

Cpl. Leo S. Parenti, APO 7488, % Postmaster, New York.

Pvt. Walter Pomianek, Brooks General Hospital, Ward 36, Fort Sam Houston, Texas.

Cpl. Barney Taylor, 46th Fighter Squadron, A. A. F., APO 459, % Postmaster, San Francisco.

Pvt. Russell Tipps, Crystal Lake, Ill.

Capt. Irwin C. Winter, M. C., Station Hospital, Flexible Gunnery School, Buckingham Air Field, Fort Myers, Florida.

MARINES

Pfc. Edward Barleak, U. S. M. C. Special Weapon Group, 8th Defense Bn., F. M. F., % Fleet Post Office, San Francisco.

Edward Ciesla, 3643 W. School St., Chicago.

Cpl. Henry Ford, Hq. and Service Btry., Anti Aircraft Gp. B. 6th Defense Btn., Navy No. 1504, % Fleet P. O. San Francisco.

John W. E. Krautter, PhM2, 25th Replacement Bn., % Fleet Postoffice, San Francisco.

NAVY

Henry H. Dohrn, A. S., Co. H., U. S. N. R., Navy V, 12 Unit, G. A. College, St. Peter, Minn.

Lieut. (jg) Charles E. Nyberg, Amphibious Training Command, N. O. B., Norfolk, Va.

Erwin Putzler, U. S. Naval Training Station, Great Lakes, Ill.

WACS

Cpl. Zona Davis, Hqs. Southwest Asia Command, APO 432, % Postmaster, New York.

Lieut. Frances Dowd, Station Hospital, Daytona Beach, Fla.

Capt. Helen Lloyd-Jones, 1550 S. U., Fort Knox, Kentucky.

Lieut. Mildred Patton, Camp Roberts, Calif.

Cpl. Zola C. Wist, The Collingwood, 45 West 35th St., New York 1.

SPARS

Angela Y. Duda, A. S., Co. 111-8-Rm. 534, Y. S. C. G. T. S., Palm Beach, Fla.

Mary Meyer, Spars Hqs., Miami Beach, Fla.

Two other A. M. A. employees have temporarily left the Association to fill specialized positions for the government for the duration. Miss Lois Stice, news editor of THE JOURNAL, is in the Office of Civilian Defense, Washington, D. C. Dr. Paul C. Barton, director of the Bureau of Investigation at these headquarters, is assistant executive officer of the Procurement and Assignment Service.

AMERICAN RED CROSS WORK IN 1943

Last year the American Red Cross experienced its busiest year in history. This organization served the armed forces abroad and at home, on the war front and in training camps, as well as civilians in many parts of the world. Industry, agriculture, other organizations and associations, as well as the individual American, enabled the Red Cross to carry on its important work.

During 1943 the American Red Cross shipped 7,405,088 food parcels to prisoners of war, weighing 8 pounds each and consisting of processed meats, cheese, powdered milk, fats and butter substitutes, dried fruits, orange concentrate, biscuits, chocolate bars, jam, dehydrated soup, hard candy, sugar, powdered coffee, soap, smoking tobacco and cigarets.

Manufacturers of drugs contributed to the success of Red Cross activities by providing the standard remedies and first aid materials used to compose the 13,000 medicine chests sent to the International Red Cross committee for distribution to prisoners of war. These chests contained standard products such as acetylsalicylic acid, soda, disinfectants, ointments, bandages, tape and cotton. The textile industry provided the necessary gauze which enabled several million volunteers to make more than 900,000,000 surgical dressings during the year. The container industry provided the cans and cartons for the individual products, as well as the fiber containers making up the complete package. This industry also provided the tin and fiber containers used in packing the 4,300,000 blood plasma units. Other branches of American industry also aided the Red Cross in supplying glass containers in which the powdered plasma and distilled water composing the unit are packed, as well as needles and tubing used in mixing the plasma with the water and administering the transfusion.

WARTIME GRADUATE MEDICAL MEETINGS

Among the subjects scheduled for early presentation under the auspices of the Wartime Graduate Medical Meetings are the following at Fort Monmouth, N. J.: Physiologic and Pathologic Background of Arthritis; Clinical Discussions and Question and Answer Period, Dr. C. W. Scull, Dr. T. F. Bach and Capt. A. R. Raynalds, February 2; Rheumatic Heart Disease, Dr. Thomas McMillan, Capt. Solomon J. Selikoff, February 9; Pathogenesis of Malaria, Dr. William Sawitz and Capt. Gerald W. Smith, February 16.

At Naval Hospital, Pa.: Obstructive Lesions of the Urinary Tract and Their Relationship in the Production of Renal Pathology, Dr. Joseph C. Birdsall, February 4.

The following program will be presented at the Wartime Graduate Medical Meetings at Percy Jones General Hospital, Battle Creek, Mich., February 14: Allergy Principles as Seen in Percy Jones General Hospital, Major Ralph I. Alford; Malaria, Its Incidence and Management, Capt. Robley D. Bates Jr.; General Consideration of Peptic Ulcer, Capt. James M. MacMillan; Some Side Lights on Soldiers with Arthritis, Major Clarence B. Whims; Presentation of Dermatologic Cases, Capt. Allen W. Pepple; Rheumatic Fever in an Army General Hospital, Major John B. McKee; Unusual Causes for Diarrhea, Lieut. Col. Charles M. Caravati; Coccidioidomycosis, Major William H. Wood Jr.; Neuropsychiatric Aspects of Fear and Its Derivatives, Lieut. Col. Paul A. Petree.

On February 21 the following program will be presented at the Wartime Graduate Medical Meetings at Percy Jones General Hospital: Amputations, Lieut. Col. Francis M. McKeever; Prostheses, Capt. Ronald M. Buck; Sciatica, Major Frank R. Mayfield; Empyema, Major Earle B. Kay; Plastic Surgery, Major Preston C. Iverson; Traumatic Perforations of Ear Drum, Lieut. Col. Charles W. Barkhorn; Penicillin in Surgery, Capt. Jose M. Ferrer Jr.; Penicillin in Gonorrhea, Major Ross M. Newman. Both meetings will be addressed by Brig. Gen. J. E. Bastion.

CASUALTIES OF U. S. ARMED FORCES SINCE OUTBREAK OF WAR

The Office of War Information reported on January 15 the number of casualties of the United States armed forces from the outbreak of the war. This total, combining the latest available War and Navy Department reports, includes 32,078 dead, 45,595 wounded, 32,478 missing and 29,707 prisoners of war. Of these 1,619 have died in prison camps, mostly in Japanese occupied territory.

The War Department report (as of Dec. 23, 1943) lists army casualties totaling 105,229. Of this number 16,831 were killed and 38,916 wounded. There were 24,067 missing and 25,415 prisoners of war. Of the wounded 20,036 have returned to active duty or have been released from the hospital. The casualties include 12,500 Philippine Scouts; of these 469 were killed and 747 wounded; the remainder are assumed to be prisoners of war.

The Navy Department (as of Jan. 14, 1944) shows casualties whose next of kin have been notified totaling 34,629, made up of 15,247 dead, 6,679 wounded, 8,411 missing and 4,292 prisoners of war.

HOSPITALS NEEDING INTERNS AND RESIDENTS

The following hospitals have indicated to the Council on Medical Education and Hospitals that they have not completed their house staff quotas allotted by the Procurement and Assignment Service:

(Continuation of list in THE JOURNAL, January 22, p. 238)

MASSACHUSETTS

Springfield Hospital, Springfield. Capacity, 285; admissions, 6,996. Dr. Eugene Walker, Superintendent (1 intern).

WISCONSIN

Milwaukee Hospital, Milwaukee. Capacity, 359; admissions, 8,472. Rev. William G. Sodt, Administrator (interns).

ORGANIZATION SECTION

A MESSAGE TO THE MEMBERS OF THE AMERICAN MEDICAL ASSOCIATION

For some ten years the physicians of the United States have been subjected to a series of stresses such as have disturbed the orderly progress of medical science in no previous period of similar length. In 1860 Oliver Wendell Holmes wrote:

> "The truth is that medicine, professedly founded on observation, is as sensitive to outside influences, political, religious, philosophical, imaginative, as is the barometer to the changes of atmospheric density."

The depression of 1929, the evolution of the Social Security Act, the first Wagner bill, the development of hospitalization and medical care insurance, the enrolment of a third of the active medical profession in the armed forces, and now the Wagner-Murray-Dingell bill represent a series of provocative crises. Each of these challenges was met by the House of Delegates, the Trustees and the officers of the Association with clearly defined statements of policy which the Association has disseminated widely. Under these policies the extension of medical service has proceeded steadily and everything possible has been done to maintain the quality of medical education and medical service at the high standard that has been our ideal. The continued pressure of the years has been climaxed by the Beveridge report, the report of the National Resources Planning Board and the introduction of the Wagner-Murray-Dingell bill. This comes when every physician not in the armed forces is giving of himself unstintedly without thought of time or physical capacity.

Today strange social philosophies pervade the radio, the press and the periodicals. Panaceas for medical problems are proffered by innumerable prescribers. Some preach distrust of medical organization, cast doubt on the loyalty of our leaders, sow dissension in our membership! These activities are no doubt a reflection of anxieties and fears. And they appear at a time when a united, loyal, solidly organized medical profession is more needed than at any previous time in our history! When our representatives appear before legislative hearings they are entitled to the loyal, enthusiastic, unified support of the constituent and component societies of the American Medical Association.

In some areas there are attempts at reorganization of the county medical society on a strictly business basis; attempts are being made to organize small groups of the states into sectional cliques; before the House of Delegates of one state a delegate actually urged a united opposition to the Southern states; here and there physicians, apparently inspired by lay employees or by the urging of outside agencies, would pour the funds of county and state medical societies, swollen by special assessments, into "public relations," as if this were some new and potent magic; there are occasional demands that the medical profession "unionize" and affiliate with one or the other of the major labor organizations. The far-seeing Oliver Wendell Holmes was right: physicians are "sensitive to outside influences, political, religious, philosophical, imaginative."

Now what are the facts? The trend of public thought is quite definitely against any such expansion of the Social Security Act as the Wagner-Murray-Dingell bill contemplates. The Council on Medical Service and Public Relations has been organized, has developed a program, has stated its policies, has secured a full time secretary, has expanded sources of information on legislative activities, is participating in public relations for the Association. The Board of Trustees has organized for suitable representation at any hearings that may be called on legislation affecting the medical profession. The publications of the Association have reached the highest peak in their history in circulation and effectiveness. A poll proves that a majority of Americans interviewed consider the American Medical Association an organization interested in the advancement of medical science, an organization devoted to the approval of that in medicine which is sound and exposing that which is fraudulent—what the experts call a "favorable symbol." And all this accomplished at a time when the employees of the Association have been reduced by one fourth by war activities or call to the armed forces, and when many others are likewise giving largely of their time to war activities.

The Board of Trustees pledges itself anew, as do the officers and employees of the Association, to do their utmost to carry out and to implement the principles, the policies and the mandates of the House of Delegates. To some 55,000 physicians who are in the armed forces the Board pledges all that the Association can do to maintain for them a medical profession free from the interference of political control. The Board is convinced that the House of Delegates will also do its utmost to hold the traditions of Americanism and American medicine inviolate until the physicians who are now with the armed forces return and themselves participate in determining the future of American medicine.

BOARD OF TRUSTEES:

Roger I. Lee, M.D., Chairman
R. L. Sensenich, M.D., Vice Chairman
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James R. Bloss, M.D.	E. L. Henderson, M.D.
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THE PHYSICIAN'S FEDERAL INCOME AND VICTORY TAX

PREPARED BY THE BUREAU OF LEGAL MEDICINE AND LEGISLATION

Federal income and victory taxpayers, last September, filed a declaration of estimated tax covering the calendar year 1943. This was the second step to put taxpayers on a current payment basis, the first step having been taken when the withholding provisions of the current tax payment act became operative on July 1, 1943. In this declaration, estimates of income and deductible expenses were made and taxes were computed on these estimates. The tax estimated to be due was either paid in full at the time the declaration was filed or one half then and the remainder on or before December 15. Persons in the armed forces on September 15 were excused from then filing the declaration.

FINAL RETURN FOR 1943

On or before March 15, a final return for 1943 must be filed. This return, in effect, will be in substantiation of the declaration that was filed in September. It will be based on the provisions of the revenue act of 1942 and of the current tax payment act of 1943. The new revenue bill now pending in Congress need not be given any consideration in filing the return. The purpose of the final return is to place the taxpayer on an actual tax basis instead of an estimated tax basis and to permit readjustments. If the taxpayer has already paid more during 1943 than his final return to be filed on or before March 15 indicates he should have paid, he will be entitled to a refund or credit. If he paid less, then he will be required to pay the difference.

TAX FORGIVENESS

In connection with this return, consideration must be given to the tax forgiveness provisions of the current tax payment act of last year. That act provided for an abatement of 75 per cent of one year's tax, or the first $50 thereof, whichever amount is the greater. The abatement is based on the lower tax of the two years 1942 and 1943 except in the case of taxpayers in service. If the 1943 tax is lower, 75 per cent of it is abated. If the 1942 tax is lower as shown on the return that was filed last March, 75 per cent of it is abated. The unabated portion will become a liability on March 15 of this year. The taxpayer may either pay the unabated portion in full at that time or he may pay one half of it at that time and the remainder on or before March 15, 1945.

If a taxpayer was in the armed forces at any time during the taxable year of 1942 or 1943, and his tax for 1942 was greater than the tax for 1943, the forgiveness will be increased to the extent that the excess of the 1942 tax over the 1943 tax is attributable to the inclusion of net income. A detailed explanation of this special provision in favor of persons in the service was included in THE JOURNAL Aug. 14, 1943, page 1134. Physicians in service should review that explanation.

THE VICTORY TAX

The method of computing the victory tax was explained in THE JOURNAL, Aug. 14, 1943. There has been one modification of the method of computation made effective since that time. Initially the revenue act conditioned the option that was accorded a taxpayer to take credit currently for the postwar credit on his having paid premiums on life insurance, purchased obligations of the United States or reduced indebtedness in an amount equal to the credit allowed. By Public Law No. 178, Seventy-Eighth Congress, approved Oct. 28, 1943, the postwar credit provisions of the revenue act were repealed and the taxpayer allowed an unconditional current credit against the victory tax as follows:

1. *Single Persons.*—In the case of a single person, a married person not living with husband or wife, or an estate or trust, an amount equal to 25 per cent of the victory tax or $500, whichever is the lesser.

2. *Heads of Families.*—In the case of the head of a family, an amount equal to 40 per cent of the victory tax or $1,000, whichever is the lesser.

3. *Married Persons.*—In the case of a married person living with husband or wife—

(*a*) if separate returns are filed by each spouse an amount equal to 40 per cent of the victory tax or $500, whichever is the lesser, or

(*b*) if a separate return is filed by one spouse and no return is filed by the other spouse, or if a joint return is filed, only one credit not exceeding 40 per cent of the victory tax or $1,000, whichever is the lesser.

4. *Dependents.*—For each dependent, excluding as a dependent in the case of the head of a family one who would be excluded for income tax purposes, an amount equal to 2 per cent of the victory tax or $100, whichever is the lesser.

If the status of the taxpayer changed during the year with respect to his marital relationship or with respect to his dependents, other than a taxpayer who uses the simplified return form, the amount of the credit will be apportioned, under rules and regulations prescribed by the Commissioner of Internal Revenue with the approval of the Secretary of the Treasury, in accordance with the number of months before and after such change. For the purpose of such apportionment, a fractional part of a month will be disregarded unless it amounts to more than half a month, in which case it will be considered as a month.

EXEMPTIONS

The revenue act of 1942 under which the final return will be filed reduced the personal exemption of single persons from $750 to $500 and of married persons or heads of families from $1,500 to $1,200. It also reduced the credits for dependents from $400 to $350. The current tax payment act of 1943 provides a special exemption for members of the armed forces, in addition to the personal exemption. The first $1,500 of the service pay of members of the armed forces, including commissioned officers, is nontaxable.

TAX RATES

The basic rate of taxation remains at 6 per cent. The surtax rate begins at 13 per cent on the first $2,000 of surtax net income and increases in rate for incomes in the higher brackets. The earned income credit of

10 per cent remains as heretofore, claimable in connection with the normal tax but not with the surtax. The pending tax bill proposes to eliminate this credit, applicable to 1944 income.

SIMPLIFIED TAX SCHEDULE

The provision for a simplified tax schedule for use by taxpayers having gross incomes of $3,000 or less, derived wholly from salaries, wages or other forms of compensation for personal services, dividends, interest, rents, annuities or royalties is continued. The use of the simplified form is optional. Generally speaking, if a taxpayer has no deductions it will be to his advantage to use this form.

WHO MUST FILE RETURNS

In General.—1. Returns must be filed by every unmarried person and by every married person not living with spouse, if gross income during 1943 was $500 or more.

2. Returns must be filed by every married person who lived with spouse, if gross income during 1943 was $1,200 or over. If both husband and wife had income and their combined gross income was $1,200 or over, they must either file separate returns or, if both are citizens or residents of the United States and if they were living together at the end of the taxable year, they may file a joint return. If a person was married and lived with spouse for only part of 1943, special rules apply with respect to the filing of returns, and physicians who come within this classification should read carefully the instructions given on the tax return blanks.

If the status of a taxpayer, so far as it affects the personal exemption or credit for dependents, changed during the year, the personal exemption and credit must be apportioned, under rules and regulations prescribed by the Commissioner of Internal Revenue with the approval of the Secretary of the Treasury, in accordance with the number of months before and after such change. For the purpose of such apportionment a fractional part of a month should be disregarded unless it amounts to more than half a month, in which case it is to be considered as a month.

Physicians in Military and Naval Service.—While a physician in the armed forces was not required to file a declaration of estimated income last September, the fact of service does not of itself excuse a failure to file the return next March. It is understood that if because of the inaccessibility of necessary records a physician in service is unable to file a complete return, he may file a tentative return on which he must estimate his income, deductions and tax as best he can and indicate on the return his reasons for following this procedure. At a later date, if that procedure is followed, a completed return must be filed and necessary adjustments in tax will be made. What has just been said relates to physicians in service who are stationed in this country.

If a physician in service is on duty outside the United States, no income tax return or payment of income tax will become due, generally speaking, until the 15th day of the 4th month following the month in which the physician ceases, except by reason of death or incompetency, to be a member of the military forces on sea duty or in service outside the continental United States, or the 15th day of the third month following the month in which the present war is terminated, whichever may be the earlier.

GROSS AND NET INCOMES: WHAT THEY ARE

Gross Income.—A physician's gross income is the total amount of money received by him during the year for professional services, regardless of the time when the services were rendered for which the money was paid, assuming that the return is made on a cash receipts and disbursements basis, plus such money as he has received as profits from investments and speculation and as compensation and profits from other sources.

If a physician receives a salary as compensation for services rendered and in addition thereto living quarters or meals, the value to the physician of the quarters and meals so furnished ordinarily constitutes income subject to tax. If, however, living quarters or meals are furnished for the convenience of the employer, the value thereof need not be computed and added to the compensation otherwise received by the physician. As a general rule, the test of "convenience of the employer" is satisfied if living quarters or meals are furnished to a physician who is required to accept such quarters and meals in order to perform properly his duties. For example, if a physician employed by a hospital is subject to immediate service at any time during the twenty-four hours of the day and therefore cannot obtain quarters or meals elsewhere without material interference with his duties and on that account is required by the hospital to accept the quarters or meals furnished by it, the value thereof need not be included in the gross income of the physician.

Net Income.—Certain professional expenses and the expenses of carrying on any enterprise in which the physician may be engaged for gain may be subtracted as "deductions" from the gross income, to determine the net income on which the tax is to be paid. An "exemption" is allowed, the amount depending on the taxpayer's marital status during the tax year as stated before. These matters are fully covered in the instructions on the tax return blanks.

Earned Income.—In computing the normal tax, but not the surtax, there may be subtracted from net income from all sources an amount equal to 10 per cent of the earned net income, except that the amount so subtracted shall in no case exceed 10 per cent of the net income from all sources. Earned income means professional fees, salaries and wages received as compensation for personal services, as distinguished from receipts from other sources.

The first $3,000 of a physician's net income from all sources may be regarded under the law as earned net income, whether it was or was not in fact earned within the meaning set forth in the preceding paragraph. Net income in excess of $3,000 may not be claimed as earned unless it in fact comes within that category. No physician may claim as earned net income any income in excess of $14,000.

PHYSICIANS IN MILITARY OR NAVAL SERVICE

Physicians in service are as much subject to the income tax law as are physicians engaged in civilian practice, except when expressly excluded from a requirement. The service pay of such physicians must be reported as income. Commutation of quarters and rental value of quarters occupied by medical officers, however, are not taxable income.

If the ability of physicians in service to pay income taxes is materially affected by such service, payment of the tax falling due before or during the service may be deferred for a period extending not more than six

months after termination of service. This deferment is authorized by section 513 of the Soldiers' and Sailors' Civil Relief Act of 1940 and applies to all members of the Army, Navy, Marine Corps and Coast Guard, and to all officers of the United States Public Health Service detailed by proper authority for duty either with the Army or Navy, on active duty or undergoing training or education under the supervision of the United States preliminary to induction into service. This does not apply to the tax imposed on employers by section 1400 of the Federal Insurance Contributions Act. This deferment is not automatic. The taxpayer must present evidence to show that his ability to pay the tax is materially impaired by reason of military service. Proof of that impairment should be submitted at the time the tax is due, on a form procurable from the offices of the collectors of internal revenue. A copy of the form was reproduced in the Feb. 28, 1942 issue of THE JOURNAL on page 737.

Any tax liability owed by a member of the military or naval forces who dies in service will be canceled, this relief being retroactive to Dec. 7, 1941. If a tax has already been assessed at the time of the death of a person in service it will be abated. If the tax has already been collected, it will be refunded as an overpayment. This tax forgiveness applies only to income taxes and not to the estate tax.

DEDUCTIONS FOR PROFESSIONAL EXPENSES

A physician is entitled to deduct all current expenses necessary in carrying on his practice. The taxpayer should make no claim for the deduction of expenses unless he is prepared to prove the expenditure by competent evidence. So far as practicable, accurate itemized records should be kept of expenses and substantiating evidence should be carefully preserved. The following statement shows what such deductible expenses are and how they are to be computed:

Office Rent.—Office rent is deductible. If a physician rents an office for professional purposes alone, the entire rent may be deducted. If he rents a building or apartment for use as a residence as well as for office purposes, he may deduct a part of the rental fairly proportionate to the amount of space used for professional purposes. If the physician occasionally sees a patient in such dwelling house or apartment, he may not, however, deduct any part of the rent of such house or apartment as professional expense; to entitle him to such a deduction he must have an office there, with regular office hours. If a physician owns the building in which his office is located, he cannot charge himself with "rent" and deduct the amount so charged.

Office Maintenance.—Expenditures for office maintenance, as for heating, lighting, telephone service and the services of attendants are deductible.

Supplies.—Payments for supplies for professional use are deductible. Supplies may be fairly described as articles consumed in the using; for instance, dressings, clinical thermometers, drugs and chemicals. Professional journals may be classified as supplies and the subscription price deducted. Amounts currently expended for books, furniture and professional instruments and equipment, "the useful life of which is short," generally less than one year, may be deducted, but if such articles have a more or less permanent value, their purchase price is a capital expenditure and is not deductible.

Equipment.—Equipment comprises property of a more or less permanent nature. It may ultimately wear out, deteriorate or become obsolete, but it is not in the ordinary sense of the word "consumed in the using."

The cost of equipment such as has been described, for professional use, cannot be deducted as expense in the year acquired. Examples of this class of property are automobiles, office furniture, medical, surgical and laboratory equipment of more or less permanent nature, and instruments and appliances constituting a part of the physician's professional outfit, to be used over a considerable period of time, generally over one year. Books of more or less permanent nature are regarded as equipment and the purchase price is therefore not deductible.

Although the cost of such equipment is not deductible in the year acquired, nevertheless it may be recovered through depreciation deductions taken year by year over its useful life, as described later.

No hard and fast rule can be laid down as to what part of the cost of equipment is deductible each year as depreciation. The amount depends to some extent on the nature of the property and on the extent and character of its use. The length of its useful life should be the primary consideration. The most that can be done is to suggest certain average or normal rates of depreciation for each of several classes of articles and to leave to the taxpayer the modification of the suggested rates as the circumstances of his particular case may dictate. As fair, normal or average rates of depreciation, the following have been suggested: automobiles, 25 per cent a year; ordinary medical libraries, x-ray equipment, physical therapy equipment, electrical sterilizers, surgical instruments and diagnostic apparatus, 10 per cent a year; office furniture, 5 per cent a year.

The principle governing the determination of all rates of depreciation is that the total amount claimed by the taxpayer as depreciation during the life of the article, plus the salvage value of the article at the end of its useful life, shall not be greater than its purchase price or, if purchased before March 1913, either its fair market value as of that date or its original cost, whichever may be greater. The physician must in good faith use his best judgment and claim only such allowance for depreciation as the facts justify. The estimate of useful life, on which the rate of depreciation is based, should be carefully considered in his individual case.

Medical Dues.—Dues paid to societies of a strictly professional character are deductible. Dues paid to social organizations, even though their membership is limited to physicians, are personal expenses and not deductible.

Postgraduate Study.—The Commissioner of Internal Revenue holds that the expense of postgraduate study is not deductible.

Traveling Expenses.—Traveling expenses, including amounts paid for transportation, meals and lodging, necessarily incurred in professional visits to patients and in attending medical meetings for a professional purpose, are deductible.

Automobiles.—Payment for an automobile is a payment for permanent equipment and is not deductible. The cost of operation and repair, and loss through depreciation, are deductible. The cost of operation and repair includes the cost of gasoline, oil, tires, insurance, repairs, garage rental (when the garage is not owned by the physician), chauffeur's wages, and the like.

Deductible loss through depreciation of an automobile is the actual diminution in value resulting from obsolescence and use and from accidental injury against which the physician is not insured. If depreciation is computed on the basis of the average loss during a series of years, the series must extend over the entire estimated life of the car, not merely over the period in which the car is possessed by the present taxpayer.

If an automobile is used for professional and also for personal purposes — as when used by the physician partly for recreation, or so used by his family—only so much of the expense as arises out of the use for professional purposes may be deducted. A physician doing an exclusive office practice and using his car merely to go to and from his office cannot deduct depreciation or operating expenses; he is regarded as using his car for his personal convenience and not as a means of gaining a livelihood. What has been said in respect to automobiles applies with equal force to horses and vehicles and the equipment incident to their use.

MISCELLANEOUS

Contributions to Charitable Organizations.—For detailed information with respect to the deductibility of charitable contributions generally, physicians should consult the official return blank or obtain information from the collectors of internal revenue or from other reliable sources. A physician may not, however, deduct as a charitable contribution the value of services rendered an organization operated for charitable purposes.

Bad Debts.—Physicians who make their returns on a cash receipts and disbursements basis, as most physicians do, cannot claim deductions for bad debts.

Taxes.—Taxes generally, either federal or state, are deductible by the person on whom they are imposed by law. Both real and personal property taxes are deductible; but so-called taxes, more properly assessments, paid for local benefits, such as street, sidewalk, and other like improvements, imposed because of and measured by some benefit inuring directly to the property against which the assessment is levied, do not constitute an allowable deduction from gross income. Physicians may deduct state gasoline taxes and state sales taxes. In some states sales taxes are imposed on the seller, but, if they are passed on to the buyer, the latter may deduct them.

State income and use taxes are deductible; federal income taxes are not. Among the federal taxes that a physician may deduct are those on admissions, dues, initiation fees, safety deposit boxes, tax on telegraph, telephone, cable and radio messages, and the federal use tax on automobiles. State automobile license fees are deductible. If a state or local fee is imposed for regulatory purposes, and not to raise revenue, the fee may not ordinarily be deducted as a tax. If such fees, however, are classifiable as a business expense, they are deductible as such. Annual registration fees imposed on physicians probably come within the category of regulatory fees and should be deducted as a business expense rather than as taxes. Local and state occupational taxes imposed on physicians are deductible either as taxes or as a business expense, depending on the purpose for which the tax is imposed.

The excise taxes imposed on employers by section 804, title VIII, and section 901, title IX, of the Social Security Act, commonly referred to as old age and unemployment benefit taxes, are deductible annually by employers in computing net income for federal income tax purposes. If the taxpayer's return is made on a cash basis, as are the returns of practically all physicians, the taxes are deductible for the year in which they are actually paid. If the return is made on an accrual basis, the taxes are deductible for the year in which they accrue, irrespective of when they are actually paid. Employees, including physicians whose employment brings them within that category, may not deduct the tax imposed on them by section 801, title VIII, of the Social Security Act, generally referred to as the old age benefits tax. If, however, the employer assumes payment of the employee's tax and does not withhold the amount of the tax from the employee's wages, the amount of the tax so assumed may be deducted by the employer, not as a tax paid but as an ordinary business expense.

Medical Expense.—A taxpayer may deduct amounts expended for medical, dental and hospital care, not compensated for by insurance or otherwise, including amounts paid for accident and health insurance, according to a prescribed formula. Deductions will be permitted to the extent that such expenses exceed 5 per cent of the net income of the taxpayer but not in excess of $2,500 in case of the head of a family, or $1,250 in case of other individual taxpayers.

Equipment Necessitated by Military Service.—The cost of equipment of an Army officer to the extent only that it is especially required for his profession and does not merely take the place of articles required in civilian life is deductible. The cost of a uniform is considered a personal expense and hence not deductible.

Laboratory Expenses.—The deductibility of the expenses of establishing and maintaining laboratories is determined by the same principles that determine the deductibility of corresponding professional expenses. Laboratory rental and the expenses of laboratory equipment and supplies and of laboratory assistants are deductible when under corresponding circumstances they would be deductible if they related to a physician's office.

Losses by Fire or Other Causes.—Loss of and damage to a physician's equipment by fire, theft or other cause, not compensated by insurance or otherwise recoverable, may be computed as a business expense and is deductible, provided evidence of such loss or damage can be produced. Such loss or damage is deductible, however, only to the extent to which it has not been made good by repair and the cost of repair claimed as a deduction.

Insurance Premiums.—Premiums paid for insurance against professional losses are deductible. This includes insurance against damages for alleged malpractice, against liability for injuries by a physician's automobile while in use for professional purposes, and against loss from theft of professional equipment and damage to or loss of professional equipment by fire or otherwise. Under professional equipment is to be included any automobile belonging to the physician and used for strictly professional purposes.

Expense in Defending Malpractice Suits.—Expense incurred in the defense of a suit for malpractice is deductible as a business expense.

Sale of Spectacles.—Oculists who furnish spectacles etc., may charge as income money received from such sales and deduct as an expense the cost of the article sold. Entries on the physician's account books should in such cases show charges for services separate and apart from charges for spectacles, etc.

NONTRADE OR NONBUSINESS EXPENSES

A new provision in the Revenue Act of 1942 permits, in the case of an individual, the deduction of all the ordinary, necessary expenses paid or incurred during the taxable year for the production or collection of income, or for the management, conservation or maintenance of property held for the production of income. While the phraseology of this provision is very broad, the Commissioner of Internal Revenue has by regulation ruled that the following expenses, among others, are not deductible under it: Commuters' expenses; expenses of taking special courses of training; expenses in seeking employment or in placing one's self in a position to begin rendering personal services for compensation; bar examination fees and other expenses incurred in securing admission to the bar, and corresponding fees and expenses incurred by physicians, dentists, accountants and other taxpayers for securing the right to practice their respective professions.

DECLARATION OF ESTIMATED TAX FOR 1944

In addition to filing the final return for 1943, individual taxpayers above the income levels described in THE JOURNAL, Aug. 14, 1943, must file another declaration of estimated tax on or before March 15, covering the anticipated tax for the calendar year 1944. This declaration, it is assumed, will be similar in form to the declaration filed last September. Forms on which it is to be filed have not as yet been distributed. They have been withheld, no doubt, because of the pendency of tax legislation in Congress that will apply to 1944 taxes.

In completing this declaration, taxpayers will follow the same procedure utilized in completing the September declaration. They must estimate their income for the year, their exemptions and deductions, and compute the tax on the basis of such estimates. One fourth of the estimated tax must be paid when the declaration is filed, except in the case of taxpayers subject to the withholding provisions of the current tax payment act. Ample provision is made for the filing of amended declarations periodically in case the original estimates are too far out of line.

When Congressional action has been completed on the pending legislation, a statement will be prepared for publication in THE JOURNAL to aid physicians in complying with its requirements.

OFFICIAL NOTES

FORTIETH ANNUAL CONGRESS ON MEDICAL EDUCATION AND LICENSURE

Program of Meetings to Be Held in Red Lacquer Room, Palmer House, Chicago, February 14 and 15

COUNCIL ON MEDICAL EDUCATION AND HOSPITALS, AMERICAN MEDICAL ASSOCIATION

MONDAY MORNING—9:30

RAY LYMAN WILBUR, M.D., *Presiding*

Introduction to the Fortieth Annual Congress.
RAY LYMAN WILBUR, M.D., Stanford University, Calif., Chairman of the Council on Medical Education and Hospitals

PROBLEMS OF POSTWAR MEDICAL EDUCATION

The Medical School Program
HAROLD S. DIEHL, M.D., Minneapolis, Dean of the Medical Sciences, University of Minnesota
Hospital Training of Medical Graduates
SAMUEL SOSKIN, M.D., Chicago, Medical Director, Dean of Postgraduate Education, Michael Reese Hospital, Chicago
Readjustments of Returning Medical Officers
WILBURT C. DAVISON, M.D., Durham, N. C., Dean, Duke University School of Medicine
Financing of Higher Education
FRED J. KELLY, PH.D., Washington, D. C., Chief, Division of Higher Education, U. S. Office of Education
Distribution of Medical Care
SAMUEL PROGER, M.D., Boston, Professor of Clinical Medicine, Tufts College Medical School

MONDAY AFTERNOON—2:00

RAY LYMAN WILBUR, M.D., *Presiding*

WARTIME PROBLEMS IN MEDICINE AND MEDICAL EDUCATION

The Army Medical Officer in Action
MAJOR GENERAL GEORGE F. LULL, M.D., Washington, D. C., Deputy Surgeon General, United States Army
Medicine in the Navy
REAR ADMIRAL ROSS T. MCINTIRE, M.D., Washington, D. C., Surgeon General, United States Navy
The Expanding Field of Public Health
THOMAS PARRAN, M.D., Washington, D. C., Surgeon General, United States Public Health Service
Medical Manpower for Civilians
HARVEY B. STONE, M.D., Baltimore, Vice Chairman, Directing Board, Procurement and Assignment Service for Physicians, Dentists and Veterinarians
Wartime Graduate Training
COMMANDER EDWARD L. BORTZ, M.D., MC-V(S), USNR, Philadelphia, Chairman, Wartime Graduate Medical Meetings.

THE FEDERATION OF STATE MEDICAL BOARDS

MONDAY—6:30

FEDERATION DINNER

Licensure Trends and Medicine
ALPHONSE M. SCHWITALLA, S.J., PH.D., St. Louis, Dean, St. Louis University School of Medicine.
Annual Report
FRANK M. FULLER, M.D., Keokuk, Iowa, President, Federation of State Medical Boards of the United States
Round Table Discussion—State Board Problems
ROOM SEVENTEEN

TUESDAY MORNING—9:30

ADAM P. LEIGHTON, M.D., *Presiding*

ACCELERATED MEDICAL TRAINING AND RELATED LICENSURE IMPLICATIONS

Premedical Training
VICTOR JOHNSON, M.D., Chicago, Secretary, Council on Medical Education and Hospitals, American Medical Association
Basic and Clinical Medical Sciences
E. M. MACEWEN, M.D., Iowa City, President, Association of American Medical Colleges
Hospital Internship
JEAN A. CURRAN, M.D., Brooklyn, Chairman, Committee on Internships, Association of American Medical Colleges
Medical Licensure Aspects
J. E. MCINTYRE, M.D., Lansing, Secretary, Michigan Board of Registration in Medicine
JAMES D. OSBORN, M.D., Frederick, Secretary, Oklahoma State Board of Medical Examiners
ROBERT R. HANNON, M.D., Albany, Secretary, New York State Board of Medical Examiners
General discussion

TUESDAY—12:30

FEDERATION LUNCHEON
ROOM NINE

TUESDAY AFTERNOON—2:00

ADAM P. LEIGHTON, M.D., *Presiding*

The Amended Nebraska Medical Practice Act
GEORGE W. COVEY, M.D., Lincoln, Secretary, Nebraska State Board of Medical Examiners
Medical Legislation
J. W. HOLLOWAY JR., Chicago, Director, Bureau of Legal Medicine, American Medical Association
General discussion
4:30 p. m. Executive Session

CENTRAL COUNCIL FOR NURSING EDUCATION

MONDAY—12:15

Luncheon for Lay Boards of Hospitals and Public Health Nursing Organizations
Address
MISS LUCILE PETRY, Washington, D. C., Director, Division of Nurse Education, United States Public Health Service.
GRAND BALLROOM

DOCTORS AT WAR

Radio broadcasts of Doctors at War by the American Medical Association in cooperation with the National Broadcasting Company and the Medical Department of the United States Army and the United States Navy are on the air each Saturday at 4:30 p. m. Eastern war time (3:30 p. m. Central war time, 2:30 p. m. Mountain war time and 1:30 p. m. Pacific war time).

The titles and guest speakers for the next four programs are as follows:

January 29.—"Dangerous Hours."
Speaker: Dr. Thomas Parran Jr., Surgeon General, United States Public Health Service.

February 5.—"White Battalions."
Speaker: Colonel Florence A. Blanchfield, Army Nurse Corps, Office of the Surgeon General, Washington, D. C.

February 12.—"Selective Placement in Industry."
Speaker: Harold A. Vonachen, medical director, Caterpillar Tractor Company, Peoria, Ill.

February 19.—"Hometown Heroes."
Speaker: James E. Paullin, M.D., President, American Medical Association.

THE CHICAGO SESSION

Applications for Space in the Scientific Exhibit

Attention is called to the fact that applications for space in the Scientific Exhibit at the Chicago session close on February 10.

Application blanks may be obtained from the section secretaries, from section representatives to the Scientific Exhibit or from the Director, Scientific Exhibit, American Medical Association, 535 North Dearborn Street, Chicago 10.

MEDICAL LEGISLATION

MEDICAL BILLS IN CONGRESS

Change in Status.—H. R. 1506 has been favorably reported by the House Committee on Military Affairs, proposing further to amend the Pay Readjustment Act of 1942. This bill, among other things, specifically provides that, in computing the pay of officers under sections 1 and 3 of the pay adjustment act, service in the Medical Reserve Corps shall be counted.

Bills Introduced.—S. 1617 proposes to enact a "Servicemen's Aid Act of 1944." It was introduced by Senator Clark of Missouri for himself and Senator George of Georgia, Senator Walsh of Massachusetts, Senator Connally of Texas, Senator Lucas of Illinois, Senator Caraway of Arkansas, Senator Vandenberg of Michigan, Senator Brewster of Maine, Senator Brooks of Illinois, Senator Wiley of Wisconsin and Senator Gurney of South Dakota. Among other things, this bill declares the Veterans' Administration to be an agency of the United States vital and essential to the successful prosecution of war, entitled to priorities second only to the War and Navy departments. The Administrator of Veterans' Affairs would be directed to expedite and complete the construction of additional hospital beds for veterans and to enter into contracts for the use of Army and Navy hospitals after the cessation of hostilities. The Administrator of Veterans' Affairs and the Secretary of War and Navy would be authorized to enter into agreements and contracts for the mutual use or exchange of use of hospital and domiciliary facilities. H. R. 3917, introduced by Representative Rankin, Mississippi, is a bill similar in objective to S. 1617. H. R. 3957, introduced by Representative Mansfield of Montana, provides for the education and training of members of the armed forces and the merchant marine after their discharge from service. H. R. 3966, introduced by Representative Brooks of Louisiana, provides that any person discharged from the active military or naval forces under a medical certificate of disability shall be classified as "IV-H" under the provisions of the Selective Service Act and shall remain in such class until eligible for reenlistment at the same rank in the branch of the military or naval forces in which he served prior to discharge. H. R. 3990, introduced by Representative Engel of Michigan, provides for vocational rehabilitation for veterans entitled to disability benefits at wartime rates. H. R. 4002, introduced by Representative Bolton of Ohio, proposes to amend the nurses' training act to provide that the head of any federal agency may request and accept transfers of student nurses transferable under the original act to a federal hospital operated by that agency in continental United States, exclusive of Alaska. H. R. 4008, introduced by Representative Fish of New York, proposes to establish at the seat of government an executive department to be known as the Department of Veterans' Security, to be administered by a Secretary of Veterans' Security appointed by the President with the advice and consent of the Senate. To this department would be transferred all functions now exercised by the Veterans' Administration.

STATE MEDICAL LEGISLATION

New York

Bill Introduced.—A. 197 proposes to provide for the establishment and administration of a system of compulsory health insurance.

Virginia

Bills Introduced.—H. 4 proposes to authorize courts to enjoin the unlawful practice of medicine, osteopathy, dentistry, chiropractic or naturopathy. No injunction may be issued with respect to a particular individual, however, until that individual has been convicted of the unlawful practice concerning which complaint is made, nor may any injunction issue until after final hearing on the merits of the case. H. 5 proposes to enact a separate naturopathic practice act and to create an independent board of naturopathic examiners to examine and license persons to practice naturopathy. "Naturopathy," according to the bill, "is defined as a system of physical culture, a drugless treatment of disease by methods supposed to stimulate or assist nature. It does not include the use of operative surgery, obstetrics, osteopathy nor the administration nor prescribing of any drug or medicine, nor the use of x-ray therapy. A certificate to practice naturopathy granted by the Board of Examiners hereinafter created will confer upon the licensee the right to practice naturopathy, to use such other sanitary and hygienic measures as are incident to this practice, and to use the title 'Doctor' or 'Dr.' in connection with his name when accompanied by the word 'naturopathy,' or to use the letters 'D.N.' in connection with his name, but the title 'Doctor' or 'Dr.' shall not be used by him alone." H. 6 proposes to authorize the creation of a so-called board of examiners in basic sciences to consist of one member of the faculty of medicine of the University of Virginia, two licensed chiropractors and one licensed naturopath. This board apparently is to examine in anatomy, histology, pathology, bacteriology, biology, diagnosis, symptomatology, sanitation and hygiene applicants for licenses to practice chiropractic or naturopathy. The bill authorizes the board to issue certificates of proficiency to those successfully passing the examinations given by it, which certificate is to authorize the holder thereof to apply to the board of chiropractic or naturopathic examiners (neither of which is now in existence) for permission to take the examination in chiropractic or naturopathy. H. 7 proposes to enact a separate chiropractic practice act and to create an independent board of chiropractic examiners. According to the bill "chiropractic is defined to be the adjustment of the twenty-four movable vertebrae of the spinal column and other articulations, and assistance of nature for the purpose of normalizing the transmission of nerve energy. It does not include the use of operative surgery, obstetrics, osteopathy, nor the administration nor prescribing of any drug or medicine. A certificate to practice chiropractic granted by the board of examiners hereinafter created will confer upon the licensee the right to practice chiropractic" and to use measures and the title Doctor of Chiropractic under stipulations similar to those appearing in H. 5 for naturopathy.

Medical News

(PHYSICIANS WILL CONFER A FAVOR BY SENDING FOR THIS DEPARTMENT ITEMS OF NEWS OF MORE OR LESS GENERAL INTEREST: SUCH AS RELATE TO SOCIETY ACTIVITIES, NEW HOSPITALS, EDUCATION AND PUBLIC HEALTH.)

ARKANSAS

District Meeting.—Dr. Gaylord F. McLeod, Magnolia, was elected president of the Fifth Councilor District of the Arkansas Medical Society at its meeting in El Dorado, January 11. Other officers include Drs. Arley D. Cathey, El Dorado, vice president and William C. Magness, Camden, Secretary. Dr. Arthur Neal Owens, New Orleans, addressed the meeting on "Some Modern Concepts of the Treatment of Burns" and Dr. Donovan C. Browne, New Orleans, "Organic Diarrhea."

CALIFORNIA

Illegal Practitioner Convicted.—Jose Manuel Gonzales Kukay, who advertised himself as "Doctor Kay" and guaranteed a thirty day cure for alcoholism by the use of capsules "and a little will power," was convicted of practicing medicine without a license, the Hollywood *Citizen-News* reported, December 29.

Personal.—Morris A. Stewart, Ph.D., Davis, associate professor of entomology at the University of California, Berkeley, has been elected president of the Pacific Coast Entomological Society. E. Gorton Linsley, Lafayette, instructor in entomology at California, has been reelected secretary.——Dr. Loren R. Chandler, San Francisco, dean of Stanford University School of Medicine, was recently honored at the dinner of the Stanford Alumni Association for his ten years on the university staff and for the work he has done on behalf of postwar medical scholarships for service men whose education was interrupted by the war.——Dr. Virginia M. Cobb, Flintridge, has resigned as medical director of the Foothill Health School for Girls, Pasadena, a position which she has held for four years.——Dr. Lillian M. Hall has recently been appointed health officer of Concord.

Lectures on Medical Jurisprudence.—On January 21 the Los Angeles County Medical Association inaugurated a series of Friday night lectures on medical jurisprudence with a talk by Dr. Frank R. Webb, Los Angeles, chief autopsy surgeon, county coroner's office, on "The Coroner's Office and the Practicing Physician." Mr. F. J. O'Ferrall, chief, division of narcotic enforcement of the state of California, gave the second lecture, January 28, on "Legal and Medicolegal Aspects of Drug Addiction." Other lecturers in the series will include:

Dr. Douglas A. Campbell, reference, state industrial accident commission, Workmen's Compensation and the Practicing Physician, February 4.

William W. Vaughn, attorney-at-law, So You're Going to Be a Witness, Doctor! February 18.

Fred N. Howser, district attorney of Los Angeles County, The District Attorney Talks to the Practicing Physician, March 3.

Hon. Samuel R. Blake, judge of superior court, Los Angeles County, The Doctor in Court, March 17.

Dr. Louis J. Regan, counsel, Los Angeles County Medical Association, Some Recent Cases—Questions and Answers, March 24.

On February 25 several of the judges of the county superior court will participate in a session entitled "Meet the Court."

COLORADO

Chamber of Commerce Opposes Wagner Bill.—The board of directors of the Denver Chamber of Commerce recently adopted resolutions opposing all the socialization features of the Wagner-Murray-Dingell bill and especially those involving the practice of medicine, according to the *Rocky Mountain Medical Journal.*

Physician Honored.—The state historical society recently awarded its first citation for distinguished historical service to the state to Dr. Margaret Long, a practicing physician of Denver since 1904 and the author of a recent book dealing with famous pioneer routes in Colorado. Dr. Long graduated at Johns Hopkins University School of Medicine, Baltimore, in 1903. According to the Denver *Post* the board of directors of the society will award these citations from time to time for outstanding historical research done in the best scholarly tradition as a means of stimulating dependable works on Colorado history.

DELAWARE

Society News.—The Delaware State Association of Clinical Laboratory Technicians was addressed on December 7 in Wilmington by J. C. Kakavas on "Penicillin—Its Production and Use." Dr. Abraham E. Rakoff, Philadelphia, addressed the November 16 meeting on "Hormone Assays and Their Diagnostic Value."

DISTRICT OF COLUMBIA

Civic Award Goes to Physician.—Dr. Charles B. Campbell, chairman of the public health committee of the Federation of Citizens Associations of the District of Columbia, has been chosen to receive the Evening Star Trophy for outstanding civic work. Dr. Campbell is a delegate to the association from the Medical Society of the District of Columbia.

The Davidson Lecture.—Dr. Walter Freeman, professor of neurology, George Washington University School of Medicine, Washington, delivered the Davidson Lecture before the Medical Society of the District of Columbia recently. His subject was "Frontiers of Multiple Sclerosis." The lecture is given on odd numbered years and commemorates the work of Dr. Edward Young Davidson. The lecturer is selected in an essay competition. This is the second time that Dr. Freeman won the honor, the first time having been in 1939, when his paper was entitled "The Surgery of Mental Disorder."

ILLINOIS

Personal.—Drs. Carl G. Johnson, Galesburg, and Andrew D. Miller, Sullivan, have recently been made members of the fifty year club of the state medical society, both having completed fifty years in the practice of medicine.

Chicago

Annual Clinical Conference.—The Chicago Medical Society will inaugurate an annual clinical conference at the Stevens Hotel, March 14-16. The program will consist of half hour periods of lectures and clinics.

University News.—Dr. Sumner L. Koch, associate professor of surgery, gave the Founders' Day Address, which opened the eighty-seventh annual session of the Northwestern University Medical School, December 29, on "The Medical Student and His Preceptors."

Dr. Warren to Give Hektoen Lecture.—Dr. Shields Warren, Boston, will deliver the twentieth Ludvig Hektoen Lecture of the Frank Billings Foundation at the Palmer House, March 24, under the auspices of the Institute of Medicine of Chicago. His subject will be "The Effects of Radiation on the Human Body."

Refresher Course in Otolaryngology.—The department of otolaryngology of the University of Illinois College of Medicine will conduct its spring refresher course, March 20-25. The course will be largely didactic, but some clinical demonstrations will be included. It is intended primarily for specialists, who, under existing conditions, are able to devote only a brief period to postgraduate review study.

Britannica Acquires Erpi Class Room Films, Inc.—Encyclopaedia Britannica, now owned by the University of Chicago, recently acquired the Erpi Class Room Films, Inc., the oldest and largest producer and distributor of motion pictures for the class room. The Citizens Board of the University of Chicago, meeting at the Chicago Club on January 24, featured one of these films on "The Digestion of Food," which clearly explains the functions and processes of the digestive system. Before the film was shown, Mr. William B. Benton, vice president of the university, gave the reasons for the acquisition of Erpi films, and Stephen M. Corey, Ph.D., executive secretary of the Britannica Educational Films Committee, explained the unusual educational advantages of moving pictures and in addition lectured on "The Digestion of Food."

MICHIGAN

Changes in Health Officers.—Dr. Georgia V. Mills, Detroit, has been appointed deputy health officer of the Saginaw City Health Unit.——Dr. James F. Wilson, Rogers City, health director of district number 4, has been appointed to a similar position with the Manistee-Benzie County health department, succeeding Dr. Lars W. Switzer, who resigned to become associated with General Motors Corporation at Bay City.

Dr. John Alexander Named Russel Lecturer.—Dr. John Alexander, professor of surgery, University of Michigan Medical School, Ann Arbor, has been selected as the Russel Lecturer at the university for the current year. The lecturer is desig-

nated each year by the executive board of the Research Club of the university, and the person chosen is one deemed to have attained the highest distinction in the field of scholarship, the honor going ordinarily to one of the senior members of the faculty. Dr. Alexander was the recipient of the Russel Award in the year 1928-1929, this award usually being given to a faculty member of the rank of instructor or assistant professor on the basis of scholarly achievement and promise for the future. Dr. Alexander is the only member of the faculty who has received both of these honors. He will deliver the Russel lecture, May 18, on "The Development of Thoracic Surgery." The winner of the Henry Russel Award will be honored at the time of the lecture.

MINNESOTA

Experiment in Rural Hospital Experience.—The School of Nursing of the University of Minnesota, Minneapolis, recently completed an experiment in rural hospital experience for senior students. The objectives of the experiment were to acquaint the students with technic and equipment and to familiarize them with some of the problems of smaller hospitals, to enable them to secure a more complete picture of patient care by seeing the background from which the patient comes and by learning of the contributions made by various groups in the community, along with their existing interrelationships, and to provide, through the students themselves, assistance to the rural hospitals in their nursing service, and to insure more adequate rural medical care by attracting more nurses to work in rural hospitals on graduation. The trial period in the experiment was divided into two terms, June 14 to July 25 and July 26 to September 5, and provided experience for four different groups of senior students. The two rural hospitals were Itasca County Hospital, Grand Rapids, and Wesley Hospital, Wadena, both having bed capacities under 50 and, in addition, 12 bassinets. In an announcement to the press, Walter C. Coffey, LL.D., acting president of the university, stated "It is expected that this successful experiment will result in its continuance at the University of Minnesota and in its widespread adoption in other parts of the nation."

MISSOURI

Fellowship in Plastic Surgery.—Dr. Earl C. Padgett, Kansas City, has made a cash donation to the Kansas University Endowment Association to be used for the establishment of a research fellowship in plastic surgery at the university's school of medicine.

NEW YORK

Dr. Godfrey Reappointed Health Officer.—Dr. Edward S. Godfrey Jr. has been reappointed as state commissioner of health by Gov. Thomas E. Dewey. *Health News* reports that the term of office is coterminous with that of governor.

Lecture on Rheumatic Fever.—Dr. John G. Fred Hiss, professor of clinical medicine, Syracuse University College of Medicine, Syracuse, gave a graduate lecture before the St. Lawrence County Medical Society, January 20, at the Potsdam Club, Potsdam, on "Rheumatic Fever—Rheumatic Heart Disease." The lecture was presented under the auspices of the state medical society and the state department of health.

New York City

New Officers of Columbia Alumni Association.—Dr. John J. H. Keating was elected president of the Alumni of the College of Physicians and Surgeons, Columbia University, at its annual dinner December 18. Other officers are Drs. Bernard S. Oppenheimer, vice president, Charles C. Lieb, secretary and James A. Corscaden, treasurer. The members of the senior class of Columbia were guests of the association at the dinner.

New Committee for Child Care.—A group of experts has been appointed by Health Commissioner Ernest L. Stebbins to work toward the raising of standards of care for children of working mothers in the four hundred day nurseries in the city. Miss Cornelia Goldsmith, B.S., on leave from Vassar College, Poughkeepsie, N. Y., where she is assistant professor of child study, is director of the study. Dr. Milton I. Levine is the pediatrician.

Fund to Study Diseases of the Mouth.—Graduates of dental colleges in the United States and abroad who have taken postgraduate work in periodontia in this country have set up a research fund to finance studies of diseases of the mouth at the College of Dentistry at New York University. Samuel M. Robbins, D.D.S., Cleveland, is chairman of the fund committee. *Science* reports that the research will be under the direction of Samuel Charles Miller, D.D.S., New York, and will cover the efficacy of various dentifrices, causes of mouth diseases and an inquiry into the incidence of trench mouth.

Students Win Awards at Commencement.—At the eighty-fifth annual commencement at the Long Island College of Medicine, Brooklyn, December 30, the following received prizes:

Dr. Howard Otto Wunderlich, Dudley Medal, for the best clinical report of a case in the medical wards of the college hospital.

Dr. Alexander Joseph Conte, Dudley Memorial Medal, for the best clinical report of a case in the surgical wards of the college hospital.

Dr. Raymond Robert Suskind, Mitchell Prize, to the best qualified in all departments of medicine.

Dr. Millard Carnrick, Ford Prize, awarded for the best dissection.

Dr. Stephen William Giorlando, Nathan H. and Johanna Szerlip Medal, for the best thesis on pneumonia.

Dr. Clarence Denton, Phi Delta Epsilon Prize, for the highest scholastic record, also the Alumni Prize, best qualified in gynecology.

Dr. Richard Lewis Bodkin, Prize of the Class of 1898, to that member of the class whose scholastic average in the fourth year has shown the greatest improvement over that of previous years.

Dr. Alexander Garcia, Obstetric Prize, for the best thesis on a subject in obstetrics.

Dr. George Edward Murray, Jeannette Millman Memorial Award, for the best essay report on some subject in the field of oncology.

Chinese Blood Bank Moves to China.—The Chinese Blood Bank, opened at 154 Nassau Street in June 1943, has concluded its trial run in New York and will be set up on the Yunnan front at the request of Lieut. Gen. Joseph W. Stilwell and Surgeon General Loo Chih-teh. The only blood bank in China, it will serve as a model and training school for similar projects on other fronts in China. During the period of its supervision by the American Bureau for Medical Aid to China in New York 1,157 persons gave blood donations, which have been made into dried plasma and are being taken to China. Forty pounds of blood bank equipment is accompanying the staff, including the Seitz filler, which makes it possible to accept the blood of donors with a history of malaria, endemic in China. Marking its new affiliation with the Chinese army, a special ceremony was held to award commissions in the Chinese army to the eight staff members of the blood bank. Miss Adet Lin, eldest daughter of Dr. Lin Yutang and secretary of the Chinese Blood Bank, was one of five young women who received commissions as first lieutenant. A Chinese born American woman, Ruth Derr, also received this commission, as did Dr. Luetta Chen, laboratory technician, Mrs. Jean Chum Liu, head nurse and Betty Eng. Dr. Chien-lung Yi, director of the Chinese Blood Bank, and Dr. C. S. Fan, bacteriologist, received majors' commissions. Louis de Fott, in charge of blood bank machinery, was commissioned captain. The young women's uniform is similar to that of the Wacs of the American army, and the men members of the blood bank wear the regulation Chinese army uniform. The technical staff of the blood bank received two years of training in the preparation and clinical use of plasma under the supervision of Dr. John Scudder, chairman of the blood bank at Presbyterian Hospital and Sloane Hospital for Women, New York, and chairman of the blood bank committee of the American Bureau for Medical Aid to China.

NORTH CAROLINA

Dr. Stevick Named Acting Director of Epidemiology.—Dr. Charles P. Stevick, Beaufort, health officer of Carteret County, has been appointed acting director of the division of epidemiology of the state board of health with headquarters at Raleigh. Newspapers report that Dr. Stevick will continue in this position for the duration.

Physician Honored.—At a meeting of the Rotary Club, December 21, Dr. Stephen A. D. Malloy was presented in absentia with a trophy naming him as "the outstanding citizen of Yanceyville" and "the first citizen of Caswell County." He was also presented with a platter on which will be inscribed "Caswell's First Citizen." In making the presentation, the Rev. Roy F. Whitley said:

For forty-five years this citizen has been a leader in the social and civic and religious life of Caswell. He served for many years on the district school committee and still is the champion of good schools. He served his county as medical examiner on the draft board during the first world war and is still rendering conspicuous service in this one. He was chairman of the Democratic executive committee for over twenty years and has been signally honored by it several times. He has been a leader in Masonry for over forty years. He is now both coroner and county physician. He has been cited by the medical fraternity for conspicuous service and honored many times. So great and successful were his untiring efforts in securing our present water system that this club called him up and presented him with a miniature spigot, which now adorns his watch chain. He has backed every forward movement in the county for the public good.

The award was presented to Dr. Malloy in absentia because he was in the hospital on account of illness.

OHIO

Dr. Bruner Completes Fifty Years in the Practice of Medicine.—Dr. William E. Bruner, professor emeritus of ophthalmology, Western Reserve University School of Medicine, Cleveland, observed his fiftieth year in the practice of medicine in Cleveland at a dinner January 4. Dr. Bruner was host at the dinner. His guests, who were his associates at the eye clinic of the old Lakeside Hospital, gave him a silver tray with their names engraved on it. Dr. Paul G. Moore, now the oldest in point of service at the eye clinics at City and University hospitals, made the presentation speech. Others present were Drs. Harley H. Brelsford, Abram B. Bruner, Webb P. Chamberlain, Homer J. Hartzell, Mark W. Jacoby, Shandor H. Monson, Michael Paul Motto, Charles F. Nelson and Harold V. Phelan. Dr. Bruner is 78 years old. He was on the staff of St. Vincent Charity Hospital until 1916, when he went to Lakeside Hospital, remaining there until after its removal to its present location on the university campus. Dr. Bruner became professor emeritus at Western Reserve in 1936.

Medicine to Benefit in Large Bequest.—Funds estimated at more than eight million dollars have been left in trust by the late Mrs. Elizabeth Severance Prentiss for the advancement of medicine, health, art, music, education and religion through institutions in which she was interested for many years before her death, January 4. Thirty-seven per cent of the assets are to support the Elizabeth Severance Prentiss Foundation, which Mrs. Prentiss established on Jan. 17, 1939 with the provision that its existence remain undisclosed until after her death. The general objectives of the foundation, for which the National City Bank of Cleveland is trustee, are to promote medical and surgical research, to initiate and further activities in the field of public health, to aid hospitals and health institutions in Cuyahoga County, to improve methods of hospital administration and to aid in support of plans to make hospitalization and medical care available to all people, especially those of low income. The remainder of the income from trust assets reserved for the public welfare is to be divided as follows: The Cleveland Museum of Art will receive 25 per cent, the Musical Arts Association 10 per cent, Western Reserve University 7 per cent, Cleveland Museum of Health and Hygiene and Oberlin College each 5 per cent, the Cleveland School of Art and the Allen Memorial Medical Library each 3 per cent, the Church of the Covenant and the Board of Foreign Missions of the Presbyterian Church each 2 per cent and Berea College, Berea, Ky., 1 per cent. In accordance with Mrs. Prentiss's wishes, a separate fund will be in trust for each of these institutions, which will receive the income from the securities in such funds. Management of the Elizabeth Severance Prentiss Foundation will be entrusted to a board of managers named by Mrs. Prentiss when she created the foundation: John P. McWilliams, Cleveland, John A. Hadden, Cleveland Heights, Lewis B. Williams, Cleveland Heights, Robert F. Bingham, Shaker Heights, and Ralph S. Schmitt, Shaker Heights. While the foundation was set up by Mrs. Prentiss as a means of devoting a part of her fortune to the interest of health and the advancement of medical science, it provides an opportunity for any one who shares her interest in those objectives to leave money for those specific purposes. In establishing the foundation she expressed the request that at least 50 per cent of the income derived from her foundation gifts be paid to St. Luke's Hospital Association as long as its standards of service and administration are on a level prevailing in a first class hospital. She also directed that most of the foundation activities be carried on in Ohio and that at least 60 per cent of all disbursement be made within the state.

OREGON

Gifts to Medical Museum.—A collection including books, pictures, diplomas and microscopes belonging to the late Dr. Andrew C. Smith has been given to the museum now being developed at the University of Oregon Medical School, Portland. Dr. Esther C. Pohl Lovejoy, New York, one of the first women graduates of the medical school and donor of the Pohl Memorial Scholarship, has given the medals and certificates of honor given her by near east countries for services during the first world war in connection with her work as director of the American Women's Hospitals, with the idea of forming a nucleus of a collection of medals and honors conferred on Oregon physicians who have been active in the present war. Dr. Albert E. Mackay, Portland, is preparing a gift of urologic instruments dating from 1887 to the present. He has also given nearly all his medical books, including a Vesalius, printed on parchment in the 16th century. Mrs. Francis Page Adams has donated a set of surgical instruments in memory of her husband, who died in 1942. The instruments were carried all through the Civil War. Other gifts include a set of instruments presented by the late Dr. Alfred Coleman Kinney, first and fiftieth president of the Oregon State Medical Society. Dr. Kinney, who died in July 1942 at the age of 93, had been named president-elect for 1950, when the society would have been 75 and he 100 years old. Dr. Kinney's son, Dr. A. W. Kinney, Ilwaco, Wash., gave a copy of Gray's Anatomy (second American edition, 1862) which had been used by his father and uncle in the dissecting room in Bellevue Hospital while they were students there, 1867-1870.

TENNESSEE

State Group Plans 1944 Meeting.—The trustees of the Tennessee State Medical Association have decided to hold their annual meeting in Nashville, April 11-13, at the Noel Hotel. The 1943 meeting was canceled because of the war, but the trustees feel sure that the meeting next year will be more than justifiable.

Postgraduate Instruction in Surgical Diagnosis.—Dr. Joseph R. Bromwell Branch, Macon, Ga., formerly professor of surgery at the National College of Medicine of Shanghai, China, has been conducting a program of postgraduate instruction in surgical diagnosis throughout the state under the auspices of the Tennessee State Medical Association. The Commonwealth Fund and other agencies are also cooperating. Subjects covered by Dr. Branch included the principles of wound healing, the management of wounds—with special reference to war wounds, the diagnosis and management of fractures including head injuries, infections of the hand illustrated with moving pictures, the treatment of burns, the management of shock, preoperative and postoperative care of patients, surgical diseases of the abdomen, acute abdominal emergencies, diseases of the breast, and nontuberculous diseases of the chest and cancer with particular reference to its prevention and early recognition.

Society News.—The Chattanooga and Hamilton County Medical Society opened its 1944 session January 6 with a banquet. The society was addressed by Dr. William D. Anderson, January 13, on "Anemias in Infancy," Dr. Cecil E. Newell, January 20, "The Treatment of Undescended Testicle," Dr. Charles R. Thomas, January 27, "Discussion of Complications of Anti-Rabies Treatment with Presentation of Case." Future meetings of the society will be addressed by the following:

Dr. D. Isbell, February 3 (subject not announced).
Dr. Chester L. Lassiter, February 10, Hemorrhage of Nose and Throat.
Dr. Gene H. Kistler, February 17, Carcinoma of the Cecum.
Dr. Oscar Merton Derryberry, February 24, Malaria Control in the Tennesee Valley.
Dr. James Lewis L. Bibb, March 2, Some New Methods in Handling the Tuberculosis Problem.
Dr. Frederick E. Marsh, March 9 (subject not announced).
Dr. A. Ellis Goodloe, March 16, Unforgetable Experiences in a Series of 529 Consecutive Bronchi and Esophageal Cases.
Drs. Samuel H. Long and William E. Van Order, March 23, Chemotherapy in Ear, Eye, Nose and Throat Disease and History and Origin of Some of the Common Drugs, respectively.
Dr. Earl R. Campbell, March 30 (subject not announced).

All physicians are of Chattanooga.

TEXAS

Meeting of Medical Examining Board.—The Texas State Board of Medical Examiners will hold its executive session and examination in Houston, March 22-24. Additional information may be obtained from Dr. Thomas J. Crowe, secretary of the board, 810 Main Street, Dallas 2.

Appointment in Bacteriology at Baylor.—Kenneth L. Burdon, Ph.D., formerly assistant professor of immunology and bacteriology at Louisiana State University School of Medicine, New Orleans, was appointed professor of bacteriology at the University of Baylor School of Medicine, Houston, recently.

WEST VIRGINIA

Refuse Osteopath's Petition for Appointment as Health Officer.—On January 7 the Supreme Court of Appeals of West Virginia refused for the second time the petition of Earl C. Hahn, D.O., St. Marys, for a writ of mandamus compelling the public health council to approve the recommendation of the Pleasants County court and appoint him health officer for the county. In the original petition filed on Nov. 30, 1943, also refused by the court, Dr. Hahn set up the fact that he is engaged in the practice of his profession at St. Marys and that he was recommended by the court of Pleasants County for appointment as county health officer for the unexpired term ending Dec. 31, 1944 and that subsequently the public health council unanimously refused to make the appointment. He charged that the sole reason the appoint-

ment was not made was that he is a doctor of osteopathy and not a doctor of medicine. Dr. Hahn claimed that, as a duly licensed osteopathic physician and surgeon, he is a "legally qualified physician" within the purview of chapter 16, article 1, section 1 of the code and as such entitled to appointment as county health officer, and that the action of the public health council in refusing to make the appointment was "arbitrary, capricious, discriminatory, prejudicial and contrary to the statutes of the state." In a supplemental petition, filed December 7, Dr. Hahn incorporates a letter from Dr. John E. Offner, state health commissioner, in which the latter is reported to have said that, under the policies of the public health council, health officers must have the same qualifications as graduates of class A medical schools and must be otherwise qualified to be licensed to practice medicine in West Virginia. Dr. Hahn claimed that, notwithstanding the fact that one of the duties of the public health council is to "define the qualifications of local health authorities and directors of divisions, and discharge other like duties," it cannot promulgate rules and regulations or make definitions which are contrary to law. He further charged that it is provided in chapter 30, article 4, section 10 of the code that osteopathic physicians and surgeons licensed in this state shall have the same rights as physicians and surgeons of other schools of medicine and that any rule or regulation or definition which deprives him of rights granted to physicians and surgeons of other schools of medicine is void and contrary to law.

GENERAL

Urology Award.—The American Urological Association announces its annual award "not to exceed $500" for an essay on the result of some specific clinical or laboratory research in urology. The amount of the prize is based on the merits of the work presented, and if the committee on scientific research deems none of the offerings worthy, no award will be made. Competitors shall be limited to residents in urology in recognized hospitals and to urologists who have been in such specific practice for not more than five years. The selected essay (or essays) will appear on the program of the forthcoming meeting of the American Urological Association, June 19-22, Hotel Jefferson, St. Louis. Essays must be in the hands of the secretary, Dr. Thomas D. Moore, 899 Madison Avenue, Memphis 3, Tenn., on or before March 15.

Ella Sachs Plotz Foundation.—Eighteen applications for grants were received during 1943 by the trustees of the Ella Sachs Plotz Foundation for the Advancement of Scientific Investigation, thirteen of which came from the United States, the others coming from five different countries in Europe, Asia and North and South America. In the twenty years of its existence the foundation has made 481 grants, which have been distributed to scientists throughout the world. According to the annual report the list of investigators, with the purpose of the research aided during the year, is as follows:

Dr. George Barkan, Boston University School of Medicine, continuation of investigations in the field of sulfonamide derivatives and related compounds.

George A. Emerson, Ph.D., West Virginia University School of Medicine, Morgantown, study of nutritional factors in susceptibility to lethal effects of anoxia.

Anna Goldfeder, New York University College of Medicine, continuation of studies on the relation between radiation effects and cell viability as indicated by induced resistance to transplanted tumors.

M. E. Hunter, Royal Victoria Hospital, Montreal, Quebec, investigation of mild nutritional deficiencies in rheumatic fever.

Hermann Lehmann, Runwell Hospital, Essex, England, continuation of research on carboydrate metabolism of isolated tissue.

Alexander Lipschutz, Santiago, Chile, South America, work on (1) the chemical structure of the steroids as related to antifibromatogenic activity, (2) the metabolic fate of fibromatogenic and antifibromatogenic steroids, (3) the fibromyoma of the prostatic region induced by estrogens in the guinea pig.

Edward P. Mumford, M.Sc., Stanford University, Calif., continuation of basic scientific research bearing on public health and the present emergency in the Pacific.

Hazel E. Munsell, Ph.D., School of Tropical Medicine, San Juan, P. R., study of the nutritional status, as regards vitamins, of sample populations of Puerto Rico.

Carl Neuberg, Ph.D., New York University, continuation of work on carbohydrate metabolism of micro-organisms and animal cells.

Herbert Silvette, Ph.D., University of Virginia, Charlottesville, investigations of the effect of low barometric pressure on renal function and on the action of drugs on the kidneys.

Thorndike Memorial Laboratory, Boston City Hospital (Dr. George R. Minot, director), in recognition of the late Dr. Francis W. Peabody's services to the foundation.

Dr. Bernhard Zondek, Rothschild Hadassah Hospital, Jerusalem, Palestine, continuation of investigations dealing with impairment and stimulation of the functions of the pituitary gland and especially of the gonadotropic hormone of the anterior pituitary lobe.

United States and American Republics Exchange Medical Knowledge.—Dr. Eugene P. Campbell, field director, division of health and sanitation, Institute of Inter-American Affairs, wrote from La Paz, Bolivia, December 22, that he wished to place correctly the credit for the great amount of work carried out in the extensive cooperative program between the United States and the American republics. THE JOURNAL, Oct. 23, 1943, page 498, carried a news item outlining the program, which, according to Dr. Campbell, was directed by Dr. Henry E. Meleney, Herman M. Biggs professor of preventive medicine, New York University College of Medicine. The time spent with the health and sanitation field party in Guatemala was under the direction of Major Robert L. Vought and in Honduras of Major Isaac Frank Tullis Jr., M. C., A. U. S., both of whom devoted much time and effort to the success of the plan. Credit is given also to the cooperation from the many physicians in the various United Fruit Company hospitals. In addition to the names published in the news item, the following are also added to the list of United States physicians who have finished or are receiving training:

James Fred Denton, Ph.D., assistant professor of bacteriology and public health, University of Georgia School of Medicine, Augusta, to Tela, Honduras.

Dr. Francis B. Johnson, professor of clinical pathology, Medical College of the State of South Carolina, Charleston, to Tela.

Dr. William C. Black, professor of pathology, University of Colorado School of Medicine, Denver, to Quiriguá, Guatemala.

Robert B. Dienst, Ph.D., associate professor of bacteriology and public health, University of Georgia School of Medicine, to Tiquisate, Guatemala.

Dr. Orlyn B. Pratt, associate professor of pathology, College of Medical Evangelists, Los Angeles, to Tiquisate.

Dr. Raymond M. Hill, instructor in medicine, College of Medical Evangelists, to Limón, Costa Rica.

Dr. Thomas H. Hunter, assistant in medicine, Columbia University College of Physicians and Surgeons, New York, to Golfito, Costa Rica.

Dr. John H. Scherer, assistant professor of medicine, Medical College of Virginia, Richmond, to Almirante, Panama.

Dr. William J. Pyles, instructor in pathology, Columbia University College of Physicians and Surgeons, to Tiquisate.

Dr. Donald S. Martin, associate professor of bacteriology, Duke University School of Medicine, Durham, N. C., to Tiquisate.

Dr. John Wyllie, professor of public health and preventive medicine, Queen's University Faculty of Medicine, Kingston, Ont., to Quirigua.

Dr. William M. Hammon, assistant professor of epidemiology, University of California Medical School, San Francisco, to Almirante.

Dr. Roger D. Baker, associate professor of pathology, Duke University School of Medicine, Buffalo, to Limón.

Dr. Henry Packer, associate professor of preventive medicine, University of Tennessee College of Medicine, Memphis, to Puerto Armuelles, Panama.

Dr. William R. Platt, assistant in pathology, Emory University School of Medicine, Atlanta, Ga., to Golfito.

Dr. Stockton Kimball, associate in medicine, University of Buffalo School of Medicine, Buffalo.

Dr. Grant O. Favorite, professor of bacteriology, Hahnemann Medical College and Hospital of Philadelphia, to Quepos, Costa Rica.

Dr. Robert F. Korns, New York State Department of Health, Amsterdam, N. Y., to Tela.

Dr. Hunter S. Cook, associate professor of pathology, Hahnemann Medical College, to Tela.

LATIN AMERICA

Professional Practice May Be Limited to Mexican Citizens.—A legislative measure, which has already passed the Mexican chamber of deputies, will, if passed by the senate, virtually deprive American professional men and women of the right to earn a living in Mexico. According to an announcement the initiative, intended to end "charlatanism" south of the border, would alter the Mexican constitution so as to deny the privilege of professional practice to all but Mexican citizens. While it would apply exclusively to the Federal District, equivalent to the United States District of Columbia and federal territories, it is viewed locally as the entering wedge for the spread of similar legislation throughout the country. Since the majority of American professional people are believed concentrated in Mexico City, which is located within the Federal District, they would be among the first to feel the weight of such restrictions. Professional classes which would be affected by the amendment include physicians and surgeons, nurses, social workers, midwives, biologists, bacteriologists, radiologists, pharmacists, pharmaceutical chemists, chemical engineers, engineering specialists, metallurgical assayers, dentists, veterinarians, agriculturists, architects, actuaries, public accountants, notary publics, lawyers, economists, primary-school teachers, professors of higher education, civilian airplane pilots and seamen.

CORRECTIONS

Medicine and the War.—In the item "Army General Praises Navy Doctor" in THE JOURNAL, January 1, page 44, the first name of Lieut. Comdr. Strongin should have been given as Herman instead of Henry.

Sublingual Administration of Drugs.—In THE JOURNAL, January 15, page 138, paragraph 2, one of the statements should read "without exception all the drugs I have studied are more readily absorbed from the sublingual area of the dog than from the same area in man." The comma between "fistula" and "tone changes" in the preceding sentence should be omitted.

Foreign Letters

LONDON

(From Our Regular Correspondent)

Dec. 18, 1943.

A New Center for Plastic Surgery: Munificent Gift by the Nuffield Trust

The gifts to medicine of Lord Nuffield, the automobile magnate, have been reported from time to time in previous letters. The latest manifestation is the offer of the Nuffield Provincial Hospitals Trust of $40,000 a year for ten years toward the cost of establishing and maintaining a plastic surgery unit by the University of Oxford. The university has accepted the offer and has appointed Mr. T. F. Kilner as the first director of the plastic surgery unit, with the title of Nuffield Professor of Plastic Surgery. The Radcliffe Infirmary will provide hospital facilities for the unit, and these will be supplemented by the Ministry of Pensions.

The total of Lord Nuffield's gifts to the University of Oxford for the development of the medical school amounts to $14,000,000. They began with the purchase of the observatory buildings and grounds adjoining the Radcliffe Infirmary, which he gave to the university to be used for the purpose of medical teaching and research. A second gift of $10,000,000 for the development of the Medical School followed. The new unit will be a center for the training of plastic surgeons and will work in close collaboration with the university laboratories, in which parallel investigations of the biochemical and other problems connected with the growth and repair of tissue, fundamental to plastic surgery, will be carried on. The need for such a center is felt. The war has greatly increased the demand for the services of plastic surgeons, and Lord Nuffield's proposal emanated from the desire to provide the best possible treatment for casualties, especially the disfigurements of burns. Although the demand may be less after the war, plastic surgery will play a great part in the services envisaged by the government for the rehabilitation of the injured.

Postwar Food Planning

Plans to take the British people off their prewar diet of "tea and margarine" and onto a diet of butter, milk and meat are being worked out for the government by dietitians. This will involve the creation of a permanent food commission with wide powers to plan the production and distribution of health supporting foods for both rich and poor. Its work will include (1) reorganization of British agriculture to increase the production of milk, eggs, fruit, vegetables and meat, (2) distribution of these foods to secure a diet adequate for health within the reach of every family, however poor, and (3) organization of food either by home production or import, if necessary, to make certain that at no time will there be a shortage of any essential food.

Food experts who have advised the government during the war are now planning for the postwar era. They hold that such a commission will revolutionize agriculture and lay the foundation of prosperity in other basic industries. White bread, margarine, jam and tea, regarded by large masses of people as a staple diet, will be discouraged. Instead, the people will be given an opportunity to increase their consumption of milk, eggs, fruit, vegetables and meat. World food policy, with which Great Britain will be closely linked, will be directed to the same end. The intention is now to extend the policy of "enough to eat" to "enough of the right kind of food." Perhaps the most valuable dietetic reform to come from wartime food control was the abolition of white bread. The deficiency of vitamins and minerals produced in "the staff of life" was one of the greatest evils of our civilization.

Control of the Use of Radon

The incautious use of x-rays for minor skin disorders by physicians without special knowledge and skill frequently resulted in burns and other injuries when radiotherapy was first introduced. Litigations followed, and the services of the Medical Defense Union were often required. In its annual report the Union now reaffirms its view that radiotherapy requires a high degree of skill if damage to normal tissues is to be avoided. The same danger exists in the use of the more recently introduced radon. The National Radium Commission and the Medical Research Council have determined the conditions under which radon, prepared from centers under their control, shall be supplied. These have also been accepted by the London Hospital Radon Service and the Manchester Radium Institute. The radon centers will supply radon only to hospitals which have been approved by the Radium Commission and not direct to individual clinicians. Only those hospitals which have a properly equipped radiotherapeutic department, with a radiotherapist and physicist as part of the staff, and which have a properly organized follow-up of patients, will be approved. Such hospitals must see that the radon is properly used, with adequate precautions against wrong dosage.

Compulsory Notification of Infective Jaundice

Because of the increasing prevalence of infective types of jaundice, which has caused the minister of health some concern, investigations have been undertaken by an expert team working at Cambridge in collaboration with the Medical Research Council. An intensive study is being made of the epidemiology of this disease, of which little is known at present, in order to bring it under control and establish methods of treatment. In order that the fullest possible information may be obtained about the incidence of jaundice, the minister of health has ordered the compulsory notification to local health officers in the eastern part of England (since the center of research is there) of the following forms of jaundice: catarrhal jaundice, acute inflammation of the liver, acute necrosis of the liver, acute yellow atrophy of the liver, toxic jaundice and infective jaundice. If the evidence received suggests an outbreak of some dimensions, the research team may have recourse to field work, which should be of assistance to health officers and practicing physicians in the diagnosis and treatment of these diseases; but it is not expected that the team will undertake field work on individual cases.

On receipt of a certificate under these new regulations or on becoming aware of a case or suspected case of one of the diseases specified, the local health officer or an officer acting under his instructions shall make inquiries and take such steps as are necessary for investigating the source of infection and preventing spread of the disease.

Marriages

HENRY O. HARDESTY, Jennings, Kan., to Mrs. Tressie B. Davidson of Sheridan, Mo., in Oberlin, Kan., December 6.

ALBERT CLIFTON SMITH JR., Glenn Springs, S. C., to Miss Rachel Ingram Monger of Lynchburg, Va., November 6.

CHARLES HERMAN ANDREWS JR., Sumter, S. C., to Miss Leila Virginia Riley of Ridgeville, November 22.

WALTER JOSEPH ALEXANDER, Binghamton, N. Y., to Miss Edith Merle Filsinger of Buffalo, October 16.

HARTFORD ERNEST GRUGAN, Lock Haven, Pa., to Miss Mary Lou Welty of Baltimore, December 25.

HENRY LEE HOWARD, Savannah, Ga., to Miss Julia T. Booker of Chapel Hill, N. C., December 16.

VINICIO G. LIVA, Lyndhurst, N. J., to Miss Helen Wagner of New York, November 23.

RICHARD B. DAVIS to Miss Nettie Schoolfield, both of Greensboro, N. C., November 27.

Deaths

John **Ralph Ballinger,** Chicago; Rush Medical College, Chicago, 1900; member of the Illinois State Medical Society and for many years chairman of the medicolegal committee; member of the House of Delegates of the American Medical Association in 1918; past president of the Northwest branch of the Chicago Medical Society; formerly assistant professor of neurology at the University of Illinois College of Medicine; member of the examining board during World War I; served on the attending staffs of the Lutheran Deaconess and Norwegian American hospitals; author of "Medico-legal Law Brief in Relation to Mal-practice Suits, with Citations"; died in Atchison, Kan., December 29, aged 68, of chronic cardiovascular renal disease, hypertension and myocarditis.

Jacob Daniel Keiper, Johnstown, Pa.; Eclectic Medical Institute, Cincinnati, 1906; member of the Medical Society of the State of Pennsylvania; past president of the Cambria County Medical Society; for many years president of the Dale National Bank; president of the Dale borough school board; served as a first lieutenant in the medical corps of the U. S. Army and as chairman of the county Selective Service Board during World War I; recently examiner for County Selective Service Board number 4; chief of the gynecology section of Memorial Hospital; died December 17, aged 60, of coronary thrombosis.

Perry Chapman Bentle ⊕ Greensburg, Ind.; Medical College of Indiana, Indianapolis, 1904; past president of the Decatur County Medical Society; served as president of the Fourth District Medical Society; school physician; on the staffs of the Decatur County Memorial Hospital and the Odd Fellows Home Hospital; member of the board of directors of the Decatur County National Bank and vice president of the board of directors of the Y. M. C. A.; examining physician for the Selective Service; member of the Rotary Club; died December 19, aged 65, of heart disease.

James Boyd Mason ⊕ London, Ky.; University of Louisville Medical Department, 1894; member of the Radiological Society of North American, Inc.; past vice president of the Kentucky State Medical Association; served as a major in the medical corps of the U. S. Army during World War I; major, medical corps, Kentucky National Guard; state surgeon for Kentucky; member of the local board of examining surgeons for pensions; examining physician for the local draft board; president of the National Bank of London; died December 2, aged 69, of chronic myocarditis.

Ursa S. Abbott ⊕ Richmond, Calif.; College of Physicians and Surgeons of San Francisco, 1902; served in the medical corps of the U. S. Army during World War I; at one time local surgeon for the Atchison, Topeka and Santa Fe Railway, the Pullman Company and the Atlas Powder Company; on the staff of the Richmond Hospital; died November 23, aged 70, of chronic myocardial degeneration.

Thomas Littleton Bauguss, Memphis, Tenn.; Memphis Hospital Medical College, 1886; died in St. Joseph's Hospital January 1, aged 92, of pneumonia, following injuries received in a fall.

Felix S. J. Bessette, Chicago; Rush Medical College, Chicago, 1889; member of the Illinois State Medical Society; died in the Mercy Hospital, December 22, aged 78, of cerebral hemorrhage.

Albert Fitzhugh Beverly, Austin, Texas; Medical Department of Tulane University of Louisiana, New Orleans, 1908; member of the State Medical Association of Texas; fellow of the American College of Surgeons; served in France and England as a major in the medical corps of the U. S. Army during World War I; died in the Seton Hospital December 9, aged 58, of general arteriosclerosis and myocarditis.

Harold Elmer Bogart, Mount Vernon, N. Y.; Cornell University Medical College, New York, 1921; assistant medical director of the New York Life Insurance Company, New York; served during World War I; died December 18, aged 47, of coronary thrombosis.

McKinley Jepthy Bohannon, Terre Haute, Ind.; Indiana University School of Medicine, Indianapolis, 1934; member of the Indiana State Medical Association; on the staff of Union Hospital, where he died December 13, aged 47, of coronary disease.

Lawrence Breitbart ⊕ Flushing, N. Y.; George Washington University School of Medicine, Washington, D. C., 1927; fellow of the American College of Surgeons; chief examining physician for local draft board number 272; associate surgeon, Flushing Hospital and Dispensary; associate visiting surgeon, Queens General Hospital, Jamaica; died December 21, aged 40, of coronary occlusion.

Horatio Alford Brown ⊕ Jackson, Mich.; Rush Medical College, Chicago, 1909; past president of the Jackson County Medical Society; fellow of the American College of Surgeons; chief medical examiner for county draft board; member of the board of managers and chief of staff, W. A. Foote Memorial Hospital; chief of staff, Mercy Hospital; died in the Good Samaritan Hospital, Lexington, Ky., December 19, aged 62, of aplastic anemia.

Ira Christopher Brown, Seattle; University of Buffalo School of Medicine, 1888; member of the Washington State Medical Association; veteran of the Spanish-American War and World War I; for many years medical director for the city public schools; died in the Veterans Administration Facility, American Lake, December 8, aged 82, of cardiac asthma.

John L. Brubaker, Altoona, Pa.; Washington University School of Medicine, Baltimore, 1874; member of the Medical Society of the State of Pennsylvania; veteran of the Spanish-American War; for many years a surgeon for the Pennsylvania Railroad; died December 13, aged 89, of pneumonia.

George Van Buskirk Buehler ⊕ Bedford, Mass.; University of Pennsylvania Department of Medicine, Philadelphia, 1895; died November 19, aged 67, of heart disease.

Leo Aloysius Bussman, Buffalo; University of Buffalo School of Medicine, 1913; died December 9, aged 58, of heart disease.

Emory Ward Carr ⊕ Lyons, N. Y.; Baltimore Medical College, 1902; formerly mayor; served as president of the school board; died in the Buffalo General Hospital, Buffalo, December 17, aged 70, of cerebral thrombosis.

Paul Bernard Cassidy ⊕ Philadelphia; University of Pennsylvania Department of Medicine, Philadelphia, 1899; for many years secretary-treasurer of the American Association of Medical Milk Commissions; served as medical director of St. Vincent's Hospital for Women and Children; on the staff of the Misericordia Hospital, where he died December 12, aged 68, of pneumonia.

Henry William Champlin, Towanda, Pa.; University of Michigan Homeopathic Medical School, Ann Arbor, 1881; died December 8, aged 86, of Parkinson's syndrome and uremia.

Benjamin Franklin Devitt, Philadelphia; Medico-Chirurgical College of Philadelphia, 1904; died in the National Stomach Hospital December 4, aged 73, of acute cardiac dilatation due to senile pneumonia.

Townsend H. Dickinson ⊕ Dayton, Ohio; Miami Medical College, Cincinnati, 1906; member of the American Academy of Pediatrics; past president of the Montgomery County Medical Society; school physician; served on the staffs of the Good Samaritan Hospital, St. Elizabeth's Hospital and the Miami Valley Hospital, where he died December 3, aged 60, of acute hemorrhagic nephritis.

William Lloyd Digges, New Madrid, Mo.; Washington University School of Medicine, St. Louis, 1897; died in St. Francis Hospital, Cape Girardeau, November 27, aged 69, of cerebral hemorrhage.

Edwin Lyon Draper ⊕ Champaign, Ill.; Harvard Medical School, Boston, 1907; Champaign County deputy coroner; served in England and France during World War I; on the staffs of the McKinley and Mercy hospitals, Urbana, and the Burnham City Hospital; member of the athletic council of the University of Illinois; died in Urbana November 29, aged 61, of cerebral hemorrhage.

Moury Irvin Ellis, East Orange, N. J.; Vanderbilt University School of Medicine, Nashville, Tenn., 1917; member of the Medical Society of New Jersey; served as a lieutenant commander at the Great Lakes Naval Training Station during World War I; on the surgical staff of the East Orange General Hospital; died December 7, aged 48, of bronchopneumonia and streptococcic throat.

John Silveira Enos, Newark, N. J.; Harvard Medical School, Boston, 1911; died November 24, aged 60, of coronary occlusion and arteriosclerosis.

Robert O. Friedrich, Milwaukee; Milwaukee Medical College, 1906; member of the State Medical Society of Wisconsin; died November 26, aged 73, of cyanide poisoning, self administered.

Maurice Jacob Greenstein, Bridgeport, Conn.; University of the South Medical Department, Sewanee, Tenn., 1905; served on the staffs of St. Vincent's and Bridgeport hospitals; died November 27, aged 61, of coronary occlusion.

Clara O. Griffin, Eden, N. Y.; University of Buffalo School of Medicine, 1906; died November 18, aged 72, of angina pectoris.

Alonzo Potter Burgess Holly, Miami, Fla.; New York Homeopathic Medical College and Hospital, New York, 1888; at one time consul for Haiti to the Bahama Islands; died November 28, aged 78, of carcinoma of the prostate.

Ernest Arthur Kaeselau ✠ Buffalo; University of Buffalo School of Medicine, 1924; member of the board, Millard Fillmore and Lafayette General hospitals; died November 27, aged 44, of coronary thrombosis following an appendectomy and umbilical hernia.

John Barney LaHiff, Clarksville, Tenn.; University of Nashville Medical Department, 1894; Vanderbilt University School of Medicine, Nashville, 1894; member of the Tennessee State Medical Association; died November 30, aged 72, of pleurisy.

Romulus Zachariah Linney ✠ Madison, Tenn.; University of Pennsylvania School of Medicine, Philadelphia, 1928; examining physician for Selective Service Board number 10 of Davidson County; member of the staff of the Protestant Hospital, Nashville; on the visiting staffs of St. Thomas Hospital, Nashville, and the Madison Rural Sanitarium and Hospital, Madison College; died in Vanderbilt Hospital, Nashville, December 19, aged 42, of carcinoma of the trachea.

Athens Vallette Lodge, Brewster, Mass.; University Medical College of Kansas City, Mo., 1900; member of the Massachusetts Medical Society; on the staff of the Cape Cod Hospital, Hyannis; died November 18, aged 71, of coronary occlusion.

Jesse S. Maloy ✠ Shinnston, W. Va.; Medical College of Virginia, Richmond, 1898; died November 21, aged 70, of carcinoma.

Wallace Jefferson Masters, Wichita Falls, Texas; University of Texas School of Medicine, Galveston, 1911; member of the State Medical Association of Texas; president of the Wichita County Medical Society in 1940; on the staffs of the Wichita General and Bethania hospitals; for many years a member of the Rotary Club; died November 26, aged 58, of coronary occlusion.

Charles Alexander McBeth, Utopia, Texas; Atlanta Medical College, 1895; served as a member of the school board of Utopia; died in the Nix Hospital, San Antonio, November 5, aged 70, of coronary thrombosis.

CAPT. JOHN E. CARTER
M. R. C., U. S. Army, 1912-1943

Charles Franklin Montgomery, Mount Pleasant, Utah; St. Louis University School of Medicine, 1906; served during World War I; died in Kansas City, Kan., November 19, aged 60, of coronary occlusion.

Paul O'Brien ✠ Rutherford, N. J.; Cornell University Medical College, New York, 1908; served in the medical corps of the U. S. Army during World War I; died in the Doctors Hospital, New York, October 19, aged 62, of carcinoma of the finger with metastases to arm, axilla and spine and terminal bronchopneumonia.

Ole Olson, Lincoln, Neb.; Nebraska College of Medicine, Lincoln, 1909; served overseas during World War I with the rank of captain in the medical corps of the U. S. Army; elected commissioner of Lancaster County in 1934; died November 8, aged 66.

William A. Paxton, Boley, Okla.; University of West Tennessee College of Medicine and Surgery, Memphis, 1907; served as mayor of Boley, assistant county health officer and chairman of the local Red Cross; died in the Park Sanitarium, Guthrie, October 15, aged about 68, of mitral regurgitation.

Wood K. Porter, Turney, Mo.; Washington University School of Medicine, St. Louis, 1894; died in St. Luke's Hospital, Kansas City, November 4, aged 73, of heart disease.

Clarence Day Powell, Valier, Mont.; College of Physicians and Surgeons of Chicago, School of Medicine of the University of Illinois, 1905; member of the Medical Association of Montana; served as mayor for three terms; died in Vancouver, Wash., November 25, aged 63, of cirrhosis of the liver.

Sergius S. Rakitin, San Francisco; Military Medical Academy, Leningrad, R. S. F. S. R., 1909; member of the California Medical Association; died in the University of California Hospital recently, aged 65, of myocardial infarction and arteriosclerotic hypertensive heart disease.

Edwin Ernest Roberts ✠ Washington, D. C.; Georgetown University School of Medicine, Washington, 1894; died November 28, aged 71.

Lisle Benjamin Robinson ✠ Atlanta, Ga.; Vanderbilt University School of Medicine, Nashville, Tenn., 1916; also a pharmacist; served with the American Expeditionary Forces during World War I; on the staffs of the Georgia Baptist, Emory, Piedmont and St. Joseph hospitals; died November 25, aged 55, of coronary occlusion.

John P. Rogers, Clarksville, Tenn.; University of Nashville Medical Department, 1907; died in the Protestant Hospital, Nashville, November 7, aged 63, of injuries received last July when a team of horses he was driving ran away.

Eugene Schreiber, Caldwell, Idaho; College of Physicians and Surgeons, Boston, 1911; member of the Idaho State Medical Association; also a pharmacist; member and past president of the chamber of commerce; died November 28, aged 54.

Otto Nicholas Schudde, Ferguson, Mo.; Barnes Medical College, St. Louis, 1898; member of the Missouri State Medical Association; served overseas in the medical corps of the U. S. Army during World War I; lieutenant colonel, medical reserve corps, U. S. Army, not on active duty; at one time affiliated with the Indian Service; formerly health officer of Franklin County; died November 29, aged 75, of myocarditis.

John L. Seabloom ✠ Red Oak, Iowa; St. Louis College of Physicians and Surgeons, 1904; served as a captain in the medical corps of the U. S. Army during World War I; died in the Bethesda Hospital, St. Paul, Minn., October 9, aged 70, of acute gastroenteritis.

Howard C. Sellards, Portsmouth, Ohio; Miami Medical College, Cincinnati, 1891; died November 27, aged 77, of heart disease.

Harvey James Skarshaug, Washburn, N. D.; State University of Iowa College of Medicine, Iowa City, 1926; member of the North Dakota State Medical Association; at one time health officer of Fargo, N. D.; died November 30, aged 45.

William Andrew Smith ✠ Mansfield, Ohio; Ohio Medical University, Columbus, 1904; served during World War I; died November 22, aged 62, of heart disease.

Elwyn Vincent Strand ✠ Bayport, Minn.; University of Minnesota Medical School, Minneapolis, 1929; for many years county coroner; member of the school board; served as health officer of the village of Lake Elmo and Bayport; on the staff of the Lakeview Memorial Hospital, Stillwater; president of the Bayport Realty Company; died November 27, aged 41, of carcinoma of the lungs.

James Alto Ward ✠ Birmingham, Ala.; Johns Hopkins University School of Medicine, Baltimore, 1918; specialist certified by the American Board of Internal Medicine; fellow of the American College of Physicians; on the staff and lecturer, Baptist Hospitals; on the staff of the Jefferson Hospital; a member of the executive committee of the board of trustees, Howard College; accidentally drowned at Inland Lake November 11, aged 51.

Myron Anson Warriner, Bridgeport, Conn.; Harvard Medical School, Boston, 1884; died December 18, aged 87.

KILLED IN ACTION

John Elliott Carter, Duxbury, Mass.; Western Reserve University Medical School, Cleveland, 1938; commissioned a first lieutenant in the medical reserve corps, U. S. Army on Jan. 14, 1941 and later a captain; posthumously awarded the Purple Heart; killed in action in the southwest Pacific area July 22, aged 31.

Correspondence

JUVENILE DELINQUENCY

To the Editor:—In the editorial December 25 concerning "Medical Aspects of Juvenile Delinquency" you fail to recognize the broader implications of the problem. Juvenile delinquency actually represents only a part of the larger problem of child guidance. When the behavior of a problem child becomes antisocial, it is then termed juvenile delinquency. When it is considered that over 10 per cent of our induction examinations and over 30 per cent of our army discharges are for neuropsychiatric causes, the more fundamental implications in childhood become obvious. Also as more and more normal men are inducted and more and more abnormal men are recognized, rejected and returned to civil life they not only constitute a serious menace to morale but very poor examples for the impressionable children.

Even in the present emergency the children who display delinquent tendencies usually were problem children and were therefore so predisposed. My own experience, contrary to your statistics, reveals that not more than a third of the children present problems because of environmental influences alone. Prior to the present emergency the figures were closer to a fourth of the total number.

The endogenous or physical-mental factors represent most of the remaining cases, although a goodly number are purely endogenous in origin.

Most delinquency is preceded by truancy. Many of these children suffer from some form of somatic disorder. This may only be an unrecognized error of refraction or an impairment of hearing. I have seen numerous "lazy children" who were actually suffering from an unrecognized cardiac, nephritic or arthritic disorder.

Maladjusted behavior is very frequently due to a neuropathologic condition that predisposes to this type of reaction. Between 1 and 2 per cent of problem children are actually suffering from an incipient psychosis.

Failure to take due cognizance of the psychologic needs of the early years of childhood predisposes to the development of various neurotic manifestations. This emotional maladjustment is too frequently expressed as delinquent behavior.

Gesell has shown how intellectual inadequacies can be recognized in infancy by developmental retardation patterns. Lurie has pioneered in pointing out the direct and indirect effect of endocrine disorders on motivation.

"The hysterical broadside because of the increase in juvenile delinquency" (to paraphrase the editorial) can be warranted if it serves to focus attention on certain needs of our children. This must be done before they develop into candidates for the half million hospital beds in mental institution or those in penal institutions or even the larger number making inadequate adjustments in civil life.

LEONARD M. DUB, M.D., Washington, D. C.

MICROMETHOD

To the Editor:—The discussion in the article by J. T. Peters published in THE JOURNAL, January 1, restricts the term micromethod to those procedures requiring the use of no more than 1 drop of blood and hence classifies its blood sulfonamide determination as a micromethod since but 1 drop of blood is used in its procedure. The volume of this drop obtained from either an ear lobe or a finger tip is described to be 0.25 cc.

The micromethod emphasizes its attractiveness by the fact that but 1 drop is necessary and yet, on closer scrutiny, that drop proves to be several drops in terms of what is generally accepted as a drop. Goodman and Gilman (The Pharmacological Basis of Therapeutics, p. 1308) state that the U. S. P. standard dropper delivers 20 drops of distilled water at 15 C., which is the equivalent of 1 cc., or 15 minims. One drop therefore becomes equal to approximately 0.05 cc. Pierce and Haenisch (Quantitative Analysis, p. 97), in their discussion on volume of drops obtainable from 50 cc. buret tips, also consider the volume of a drop to be approximately 0.05 cc.

If the term "drop" is permitted to be used in a quantitative sense, limits of its volume ought to be agreed on and universally recognized.

HAROLD M. COLEMAN, PH.D., Chicago 15.

EKG OR ECG

To the Editor:—In THE JOURNAL for Dec. 25, 1943 Dr. E. B. Krumbhaar objects to the use of EKG and makes propaganda for ECG. Dr. Krumbhaar is mistaken. EKG is the right way of spelling. E K G are the initials of the three Greek words elektron, kardia and gramma; kardia means heart, as we can find in the gospel of St. Matthew, 15:8, in Novum Testamentum Graece, by Nestle, St. Louis, Concordia Publishing House, 1942. Surely St. Matthew knew how to spell kardia.

HUGO STANKA, M.D., Anna, Ill.

[On receiving a copy of Dr. Stanka's letter, Dr. Krumbhaar replied:]

To the Editor:—My suggestion of course assumed, correctly, I believe, that the word to which the abbreviation ECG referred had long since become Anglicized. "Electrokardiogram" is not found in our medical dictionaries any more than "kardiology," "kardiac," "kyst," "kakexia," "kakoethes arguendi" or numerous other words derived originally from the Greek.

As to the line about St. Matthew, I understand that it is not known whether Matthew knew only Aramaic or Greek as well. It reminds one of the good old fundamentalist who insisted on retaining the King James version of the Bible, as opposed to the Authorized, because "the language that was good enough for St. Paul is good enough for me"!

E. B. KRUMBHAAR, M.D., Philadelphia.

VISUAL TEST FOR MALINGERING

To the Editor:—From experience gained partly in induction work, where the factors of limited time and space and the absence of any elaborate equipment are the rule, it is suggested that the use of the so-called naval or "approach" method of recording vision is deserving of wider application as a quick and reliable test for malingering.

In the execution of this method the person under examination is directed to approach a Snellen test chart placed at a distance of 20 feet. If now the examinee insists he cannot read for example beyond the 70 foot type at this distance (20/70 representing a fraction with 20 feet as the numerator and 70 feet as the denominator) and is able at a distance of 10 feet to read the line indicating the 20 foot type (10/20 a fraction having 10 feet as the numerator and 20 feet as the denominator) he is obviously malingering for, by conversion, 10/20 is the equivalent of 20/40, and in each instance a corresponding angle of 10 minutes of arc is subtended.

With a conversion formula (*Arch. Ophth.* **30**:138 [July] 1943) and a rationale for the "approach" method of designating vision (*ibid.* **30**:377 [Sept.] 1943) available the problem of interpreting and evaluating Snellen symbols is more clearly defined and by the same token affords additional proof of the soundness of conclusions previously outlined.

J. A. C. GABRIELS, M.D., Albany, N. Y.

SUBSTITUTE FOR TALCUM POWDER IN SURGERY

To the Editor:—In THE JOURNAL, Dec. 11, 1943, Seelig, Verda and Kidd recommended potassium bitartrate as a substitute for talcum powder in surgery.

Some time ago we commenced a series of experiments using a substitute for talc. The first compound used was starch, selected because of its smoothness and qualities as a dusting agent, as well as its being a carbohydrate which might be broken down into its simpler components in the peritoneal cavity and be utilized. We found, however, that starch could not be sterilized in the usual manner without becoming gelatinized and forming a glaze over the gloves causing them to be stiff and stick together. Despite this it was used for two abdominal operations on one dog, the first consisting in simply handling the bowel with unrinsed gloves, and the second rubbing a small quantity (several grams) on the bowel surface. In neither operation were adhesions formed; no traces of the powder could be found. However, it was discarded because of its inability to withstand proper sterilization.

At this time, while searching for another talc substitute, the Sept. 11, 1943 issue of THE JOURNAL contained a communication by Seelig stating that he and his associates had given up the use of starch, which he had recommended in THE JOURNAL, April 17, 1943, and were using potassium bitartrate instead and had found it satisfactory. We decided to confirm their findings.

Trial sterilization of the gloves powdered with potassium bitartrate showed that it withstood the sterilization without change and was satisfactory physically. As yet we have noticed no appreciable shortening in the life of the rubber gloves but have done no specific experiments to determine this. No skin sensitivity to the bitartrate has yet appeared.

To date we have performed thirty-four abdominal operations on 10 dogs (sterile procedure). Thirty-four persons have scrubbed and used gloves prepared with potassium bitartrate three times each. In all of these operations the powder was not washed off of the gloves, and in one several grams of the powder was rubbed onto the bowel surface, and in none of these were adhesions found which could be attributed to the powder, nor could traces of the powder be found. None of the dogs showed any toxic manifestations. In several dogs that have been studied post mortem three to four weeks after the fourth operation (abdominal) by second year students, the abdomen has been entirely free of adhesions. This is unusual, since inexperienced students in their first contact with surgery usually handle the bowel excessively. The results obtained with potassium bitartrate are even better than those obtained when using the "wet glove technic," in which no powder is used.

Thus the findings of our small series can be summarized as follows:

1. Starch, although it did not produce adhesions in the peritoneal cavity, was not able to withstand sterilization without undergoing change.

2. Potassium bitartrate intraperitoneally does not cause adhesions.

3. Potassium bitartrate causes no harmful effects even from the absorption of several grams of powder.

4. No skin sensitivities have been found in those using potassium bitartrate as a dusting powder.

The foregoing findings are thus seen to confirm those of Seelig, Verda and Kidd on the use of potassium bitartrate as a surgical dusting powder.

DANIEL C. RIORDAN, M.D.,
Surgical Laboratory, Stanford University
School of Medicine, directed by
F. L. Reichert, Professor of Surgery.

AMINOPHYLLINE DEATHS

To the Editor:—In an article on "Aminophylline Deaths" (THE JOURNAL, Dec. 25, 1943, p. 1115) Dr. G. A. Merrill reported 3 deaths during or shortly after intravenous injections of 0.25 Gm. of aminophylline. The impression is given that this drug is very dangerous in cases of cardiac failure and in bronchial asthma. Two of these patients had heart disease (coronary and cardiac decompensation). The third patient was 73, with a blood pressure of 200/90, pulse rate 120, and heart sounds of poor quality. The diagnosis of bronchial asthma was made, but it is obvious that hypertensive cardiovascular disease also existed. Therefore all 3 patients had heart disease.

No one will question the possible danger of intravenous therapy in acute heart disease, especially acute coronary occlusion. The intravenous use of aminophylline in heart disease has not met with universal approval and it undoubtedly has its dangers; fatalities from its use do occur.

But the intravenous administration of aminophylline in bronchial asthma, given slowly, is practically free from danger. I have given many hundreds of these injections at Wesley Memorial Hospital, in the office and in the home and have yet to see any dangerous reaction beyond an occasional and temporary episode of nausea. The drug, when given by vein, quickly helps almost all patients with bronchial asthma and is especially indicated for an asthmatic patient who has had too much epinephrine.

The intravenous use of aminophylline should therefore be continued in cases of uncomplicated bronchial asthma.

LEON UNGER, M.D., Chicago.

Medical Examinations and Licensure

COMING EXAMINATIONS AND MEETINGS

BOARDS OF MEDICAL EXAMINERS
BOARDS OF EXAMINERS IN THE BASIC SCIENCES

Examinations of boards of medical examiners and boards of examiners in the basic sciences were published in THE JOURNAL, Jan. 22, p. 257.

NATIONAL BOARD OF MEDICAL EXAMINERS

NATIONAL BOARD OF MEDICAL EXAMINERS: *Part III.* Various centers, January. Sec., Dr. J. S. Rodman, 225 S. 15th St., Philadelphia.

EXAMINING BOARDS IN SPECIALTIES

AMERICAN BOARD OF ANESTHESIOLOGY: *Oral. Part II.* Chicago, June 12-16. Final date for filing application is March 12. Sec., Dr. Paul M. Wood, 745 Fifth Ave., New York.

AMERICAN BOARD OF DERMATOLOGY AND SYPHILOGY: *Written.* Various large cities, May 8. *Oral.* Chicago, June 9-10. Final date for filing application is April 1. Sec., Dr. C. Guy Lane, 416 Marlboro St., Boston.

AMERICAN BOARD OF INTERNAL MEDICINE: *Written.* Various centers, Oct. 16. Candidates in military service may take examination at their place of duty. Final date for filing application is August 15. Asst. Sec., Dr. W. A. Werrell, 1301 University Ave., Madison, Wis.

AMERICAN BOARD OF OBSTETRICS & GYNECOLOGY. *Written. Part I.* Various centers, Feb. 12. Candidates in military service may take Part I at their place of duty. *Oral. Part II.* Pittsburgh, June 7-13. Sec., Dr. Paul Titus, 1015 Highland Bldg., Pittsburgh.

AMERICAN BOARD OF OPHTHALMOLOGY: Chicago, Oct. 5-7. Sec., Dr. S. Judd Beach, P. O. Box 1940, Portland, Me.

AMERICAN BOARD OF OTOLARYNGOLOGY: *Oral.* New York City, June 1-4. Sec., Dr. Dean M. Lierle, University Hospitals, Iowa City, Ia.

AMERICAN BOARD OF PEDIATRICS: *Written.* Locally, Feb. 4. *Oral.* Philadelphia, March 25-26, and San Francisco, May 6-7. Sec., Dr. C. A. Aldrich, 115½ First Ave. S.W., Rochester, Minn.

AMERICAN BOARD OF PSYCHIATRY AND NEUROLOGY: *Written.* Various centers, March 31. *Oral.* Philadelphia, May 12-13. Final date for filing application is Feb. 29. Sec., Dr. Walter Freeman, 1028 Connecticut Ave. N.W., Washington, D. C.

AMERICAN BOARD OF UROLOGY: *Oral.* Chicago, Feb. 15-17. Sec., Dr. Gilbert J. Thomas, 1409 Willow St., Minneapolis.

Bureau of Legal Medicine and Legislation

MEDICOLEGAL ABSTRACTS

Dental Practice Acts: Deceptive Advertising by Operator of Chain of Dental Offices.—Campbell, who was licensed to practice dentistry in Washington and in California, conducted a chain of twelve or more dental offices, one of which was in Seattle and the rest in California. At his Seattle office licensed dentists, resident in the state of Washington, were employed by him and conducted by far the greatest part of the practice of dentistry carried on there. Campbell visited his Seattle office about once every two months, and only on those infrequent occasions was he present in the state or did he personally practice dentistry in the state. Proceedings to revoke his license to practice dentistry in Washington were instituted on the ground that in the conduct of his Seattle office he used false and misleading advertising the tendency of which was to deceive the public into believing that he was personally doing the things necessary to be done as a part of the practice of dentistry in that office. From an order of the director of licenses revoking his license Campbell appealed to the superior court for Thurston County, which, after hearing the matter de novo on the record before it, in effect affirmed the order of the director, and Campbell appealed to the Supreme Court of Washington.

Campbell's principal contention was that the trial court erred in authorizing the cancellation of his license. The court then discussed in detail the evidence adduced at the original hearing. Aside from the stipulation between counsel, said the court, that evidence consisted entirely of a series of advertisements which Campbell periodically had inserted in various newspapers in Seattle and which the state contends deceived or tended to deceive the public. In a prominent position in these advertisements appeared Campbell's picture, immediately above which were the words "Dr. Campbell says," and to the side of it was a circular diagram containing the exordium: *"I prefer that you use my* No Money Down *Plan instead of paying cash."* The words which are italicized were in longhand style, while the remainder of the advertisement was in heavy black type. The advertisements were so arranged with reference to the picture as to make it appear that the words therein were being uttered by Campbell. At the bottom of the advertisements was the announcement "Dr. J. C. Campbell, Dentist, 1520 Westlake." The question then is, continued the court, whether or not these advertisements contravene the provisions of the dental practice act that make it unlawful for any person "in any way to advertise in print any matter with a view of deceiving the public, or in any way that will tend to deceive or defraud the public." Campbell's purpose in inserting these advertisements in the public press was, of course, to attract the attention of the public and thereby to solicit its patronage. The advertisements, it will be observed, are highly personalized. First, there is the picture of Campbell. Then, the individual thus pictured is represented as speaking personally and directly to the reader. Then at the conclusion of the advertisement is the name of the individual who has thus spoken. The advertisements, taken as a whole, convey the idea that Campbell is an independent practitioner, not only owning the dental office but actually and personally conducting or immediately supervising its entire operations. There is no mention or indication of the fact that the office is but one of twelve or more similar offices, all except this particular one being in the state of California and requiring a corresponding amount of Campbell's time and attention. There is no suggestion that the Seattle office constitutes a clinic, company or an association of dentists. For aught that appears in the advertisements, the office is operated and conducted personally by a single dentist. The reader of the advertisement is induced or invited to believe that, if he responds thereto, he will receive Campbell's personal attention. At least it can be said that such advertisements "will tend to deceive or defraud the public" in that respect. As was stated in *Campbell* v. *State*, 12 Wash. (2d) 459, 122 P. (2d) 458; J. A. M. A. **119**:584 (June 13) 1942, wherein was involved the right of this same dentist to open and conduct another dental office in the city of Tacoma under his general system of operation:

> The relationship between dentist and patient is inherently personal in the highest degree. Certainly it is within the province of the legislature to protect the public against all forms of fraud and deception tending to conceal the professional identity of the dentist who is, in fact, rendering the service in the particular office frequented by one in need of dental assistance. By the practice denounced by the act [the Washington dental practice act] the public may be unwittingly deprived of a personal relationship which may rightfully be expected, and another personality substituted therefor. The use of the name of a certain dentist as conducting an office for the practice of dentistry should mean something more than merely physical ownership of the office, or the right to use the name, which may have a value because of long continued use or commercial advertising.

The trial committee that acted for the director of licenses in the original hearing and the superior court for Thurston County on a rehearing of the matter concluded that these advertisements had a tendency to deceive the public. We accept those findings and conclusions as correct.

Campbell next argued that the charges against him have no foundation in fact because there was no evidence that anybody was actually deceived by the advertisements referred to. Manifestly, answered the court, it would have been practically impossible for the state to ascertain and interview the many persons who may have visited Campbell's office in response to his newspaper advertising. It was not incumbent on the state to produce such evidence. In any event, the advertisements speak for themselves and reveal their own peculiar tendency to deceive the public in the respect referred to.

Finally, Campbell contended that the provisions of the dental practice act under which the proceedings against him were instituted are unconstitutional because of ambiguity, vagueness and uncertainty. Whatever may be the weight on this question of out of state authorities cited by Campbell to sustain his contention, said the court, we need go no further than to consult our own decisions in Washington in order to uphold the constitutionality of the statute under consideration. In *State Board of Medical Examiners* v. *Jordan*, 92 Wash. 234, 158 P. 982, this court dealt with a statute that made unprofessional conduct a ground for revoking a license to practice medicine and which, in defining such misconduct, included the following: "Third. All advertising of medical business which is intended or has a tendency to deceive the public or impose upon credulous or ignorant persons, and so be harmful or injurious to public morals or safety." It was contended there, as it is here, that the statute was uncertain, furnishing no standard by which the propriety or impropriety of questioned conduct could be measured. This court there said:

> The one [provision of the statute quoted above] relating to advertisement cannot well be made more specific. To describe in express terms a faulty advertisement is practically to instruct the defendant how to evade it, and as to the limitless variations of language, symbols and verbal or pictorial allurements, no human ingenuity could possibly anticipate and forestall them.

Again, in *State Board of Medical Examiners* v. *Macy*, 92 Wash. 614, 159 P. 801, the same statute involved in the Jordan case was held not to be "void or unconstitutional because of its vagueness or uncertainty." We hold, continued the court, that the provisions of the dental practice act, in effect, authorizing revocation of a license of a dentist advertising in print any matter with a view of deceiving the public, or in any way that will tend to deceive or defraud the public, are not unconstitutional because of ambiguity, vagueness or uncertainty.

The order of the director revoking Campbell's license to practice dentistry in Washington was affirmed.—*In re Campbell, 142 P. (2d) 492 (Wash., 1943).*

Society Proceedings

COMING MEETINGS

Annual Congress on Industrial Health, Chicago, February 15-16. Dr. Carl M. Peterson, 535 N. Dearborn St., Chicago 10, Secretary.

Annual Congress on Medical Education and Licensure, Chicago, February 14-15. Dr. Victor Johnson, 535 N. Dearborn St., Chicago 10, Secretary.

New Orleans Postgraduate Medical Assembly, New Orleans, March 6-9. Dr. Joseph S. D'Antoni, 1430 Tulane Ave., New Orleans 13, Secretary.

Society of University Surgeons, Nashville, Tenn., February 10-12. Dr. Alexander Brunschwig, 950 East 59th St., Chicago, 37, Chairman, Program Committee.

Current Medical Literature

AMERICAN

The Association library lends periodicals to members of the Association and to individual subscribers in continental United States and Canada for a period of three days. Three journals may be borrowed at a time. Periodicals are available from 1934 to date. Requests for issues of earlier date cannot be filled. Requests should be accompanied by stamps to cover postage (6 cents if one and 18 cents if three periodicals are requested). Periodicals published by the American Medical Association are not available for lending but can be supplied on purchase order. Reprints as a rule are the property of authors and can be obtained for permanent possession only from them.

Titles marked with an asterisk (*) are abstracted below.

American Journal of Public Health, New York

33:1309-1386 (Nov.) 1943

Immediate Wartime Outlook and Indicated Postwar Conditions with Respect to Control of Venereal Diseases. T. B. Turner.—p. 1309.
*Fatal Staphylococcic Intoxication from Goat Milk. L. A. Weed, A. C. Michael and Rolla N. Harger.—p. 1314.
Sanitary Privy and Its Relation to Public Health. E. S. Tisdale and C. H. Atkins.—p. 1319.
Further Studies on One Type of Paracolon Organism. C. A. Stuart and R. Rustigian.—p. 1323.
Neutralization of SK Murine Poliomyelitis Virus and of Theiler's Virus of Mouse Encephalomyelitis by Human Sera. E. Seligmann and C. W. Jungeblut.—p. 1326.
Wartime Health Education in Secondary Schools. Ruth E. Grout.—p. 1333.
Health Education in Action. A. Oppenheim.—p. 1339.
Postwar Malaria Prevention by County Health Department. W. N. Sisk.—p. 1343.
Removal of Poliomyelitis Virus from Sewage by Activated Sludge Process and the Separation from Sludge of an Antibacterial and Antiviral Substance. H. J. Carlson and C. F. McKhann.—p. 1347.
Public Health Significance of U. S. Cadet Nurse Corps. Lucile Petry.—p. 1353.

Staphylococcic Intoxication from Goat Milk.—Weed and his associates present epidemiologic data on a familial outbreak in which 3 patients developed food intoxication by drinking milk of a goat suffering from an acute suppurative mastitis due to Staphylococcus aureus. Two of the patients (3 and 4 years old) died within twenty-four hours after each drank ¼ pint (250 cc.) of the milk. Individuals who consumed the cooked milk did not develop symptoms. The goat died within twenty-four hours after the milking related to the onset of symptoms. Material taken from the udder post mortem reproduced the symptoms in kittens. The evidence suggests the possibility that if an enterotoxin was present it was thermolabile.

Annals of Surgery, Philadelphia

118:761-920 (Nov.) 1943

*Surface Treatment of Burns: Comparison of Results of Tannic Acid, Silver Nitrate, Triple Dye and Vaseline or Boric Ointment as Surface Treatments in 150 Cases. G. H. A. Clowes Jr., C. C. Lund and S. M. Levenson.—p. 761.
*Gunshot Wounds of Abdomen: Survey of 238 Cases. D. C. Elkin and W. C. Ward.—p. 780.
Principles Underlying the Operative Approach to Treatment of Myocardial Ischemia. C. S. Beck.—p. 788.
Clinical Appraisal of the Beck Operation. H. Feil.—p. 807.
Total Gastrectomy by the Transthoracic Approach: Report of 7 Cases. R. H. Sweet.—p. 816.
Carcinoma of Duodenum: One Stage Radical Pancreaticoduodenectomy, Preserving the External Pancreatic Secretion: Case Report. C. G. Child.—p. 838.
New Concept Regarding Origin of So-Called Primary Carcinoma of Hyperplastic Thyroid. E. Goetsch.—p. 843.
Carcinoma of Breast: II. Criteria of Operability. C. D. Haagensen and A. P. Stout.—p. 859.
*Immediate Effects on Renal Function of Onset of Shock Due to Partially Occluding Limb Tourniquets. A. C. Corcoran, R. D. Taylor and I. H. Page.—p. 871.
Primary Osteogenic Sarcoma of Bladder: Complete Review of Sarcomas of Bladder. A. R. Crane and R. G. Tremblay.—p. 887.
Meningioma: Case Report. C. Pilcher.—p. 909.
Anterior Sacral Meningocele: Report of 3 Cases. F. P. Shidler and V. Richards.—p. 913.

Surface Treatment of Burns.—Clowes and his associates studied 150 cases of burns, comparing the results following surface applications of ointment gauze dressings, tannic acid-silver nitrate and triple dye. There were two slight clinical infections in the 75 patients with second degree burns and no fatalities. Edema of the face disappeared rapidly without application of pressure. Skin healing of second degree burns of all areas except the back took place a few days earlier if ointment of triple dye was used than if tannic acid was used. Skin healing of second degree burns of the back took place a few days earlier if petrolatum gauze or boric acid ointment was used for treatment than if either tannic acid-silver nitrate or triple dye was used. Skin healing of second degree burns of the face was faster and of the back slower than of all other parts of the body. Fifty-seven patients had mixed second and third degree burns with 9 per cent or less of body area involved in third degree burn. There were 19 instances of clinical infection among these patients, but none died. Eighteen patients with more than 10 per cent of surface area involved by a third degree burn either died or were seriously ill. Two died of shock or other complications within one and five days respectively. All 5 patients with 10 or 15 per cent of third degree burns survived, while only 3 of 11 with 20 per cent or larger areas survived. The statistics suggest, but do not prove, that petrolatum gauze and triple dye treatment were safer for large third degree burns than treatment with tannic acid-silver nitrate. Areas treated with petrolatum gauze became free from slough and were grafted earlier than areas treated with the other two methods.

Gunshot Wounds of the Abdomen.—Elkin and Ward report observations on 238 patients admitted between 1937 and 1943 to the surgical service of the Emory University Hospital with the provisional diagnosis of gunshot wound of the abdomen. All the patients were Negroes. There were 29 shotgun wounds, while 209 were produced by rifle or pistol. Seventeen patients died immediately after admission, and 4 died on the operating table. There were 94 postoperative deaths among 199 patients operated on. Thirty-three of the operative group were given sulfanilamide. Fourteen received the drug subcutaneously and 19 received it both subcutaneously and intraperitoneally; the number of deaths was 6 and 5 respectively, making a mortality of 33 per cent. The drug seemed to be of considerable value in controlling the peritonitis. Wounds of entrance and exit were excised, treated with sulfanilamide crystals and packed. The contents of the peritoneal cavity were aspirated. Usually the cecum was examined first; the small intestine was then inspected from the ileocecal junction to the ligament of Treitz. Perforations were repaired as they were found. When resection was necessary, clamps were placed on the open ends as a landmark while the remainder of the small intestine was examined. The large intestine was next explored, and after that the stomach and retroperitoneal structures. Moderate size retroperitoneal hematomas were left undisturbed unless they involved the kidneys, pancreas or duodenum. Minor lacerations of the kidney or spleen can usually be sutured; otherwise these organs should be removed. Most wounds of the liver were not bleeding at the time of exploration. Perforations of the diaphragm (on the left) should always be closed to avoid the possibility of herniation. The mortality rate in gunshot wounds of the abdomen has remained high and will probably continue so. The severity of the wound, the time required to reach the hospital and the physical status of the patient are uncontrollable factors. Type of anesthesia, prompt and complete exploration, with repair of damage, proper use of chemotherapy and postoperative management are factors which are controllable and important.

Effects on Renal Function of Shock Due to Limb Tourniquets.—According to Corcoran and his associates, changes of renal blood flow and function during shock are of current interest since they may help explain the pathogenesis of renal failure in "traumatic anuria" (crush syndrome). The authors describe variations in renal hemodynamics and function which may occur during the onset of shock in anesthetized dogs after renal denervation, after splenectomy and during transfusion of blood. The choice of partially occluding limb tourniquets as a method of producing shock was made because of its simulation of the conditions under which clinical traumatic anuria may occur and because it permits continuous observation of systemic changes. Each experiment consisted of a control period of thirty minutes of observation, following which partially occluding limb tourniquets were applied. The observations were continued for about four hours, when the tourniquets were removed and the experiment continued for another eighty minutes or more. Some of the animals were killed at the end

of this time, but the majority died eight to fourteen hours after the application of the tourniquets. The observations made during the experiments include the renal clearances of diodrast and inulin, urine volume, arterial pressure and hematocrit index (method of Wintrobe). The authors found that the onset of shock due to the four hour application of partially occluding hind limb tourniquets to anesthetized dogs causes a definite depression of renal function, which is the result of reduction in renal blood flow. Hypotension is only a minor and inconstant element in this decrease in renal blood flow; increased renal resistance is much more important. Among the causes of increased renal resistance, blood viscosity due to a rise of hematocrit index is not significant, for splenectomy, which prevents the increase of hematocrit, does not interfere with the reduction of renal blood flow. Renal vasoconstriction too accounts for only a small proportion of the renal ischemia which follows tourniquet application and release, as is shown by renal denervation. The decrease in renal blood flow seems to be due to some humorally circulating vasoconstrictor substance. Such a material was demonstrable in the plasma of several animals in which its presence was associated with increased renal resistance, predominantly at the glomerular efferent arterioles. With injury, as after the release of tourniquets, the diodrast renal extraction ratio may be depressed, so that normal interpretations cannot be given to changes in plasma diodrast clearance and filtration fraction. The renal vasoconstriction associated with the onset of shock serves to explain transient signs of renal irritation. Although it may play a part in the genesis of "traumatic anuria" ("crush syndrome") it alone does not reproduce this state.

Archives of Dermatology and Syphilology, Chicago

48:479-578 (Nov.) 1943

Histopathologic Study of Psoriasis. J. W. Burks and H. Montgomery.—p. 479.
Bubonulus in Granuloma Inguinale. N. Sobel and N. Pensky.—p. 494.
Inhibitory Effect of Monochlormercuricarvacrol on Growth of Pathogenic Fungi. C. E. Georgi.—p. 497.
*Treatment of Tinea with Ethyl Chloride. N. Bograd.—p. 511.
Schematic Classification of Animal Organisms Causing Dermatologic Diseases. M. E. Obermayer.—p. 512.
Results of Questionnaire on Patch Tests: Analysis of Answers by Two Hundred and Eleven Dermatologists and Fifty-Five Industrial Physicians. J. G. Downing.—p. 514.
Vitiligo in Three Siblings. K. Jaenicke.—p. 519.
Congenital Auricular Fistula. F. T. Becker.—p. 520.
*Fixed Eruption, Conjunctivitis and Fever from Sulfathiazole. W. Director.—p. 523.
CII. Sporotrichosis Following a Mosquito Bite: Description of Lesions in Girl of Indian and French Descent. M. Moore and G. Manting.—p. 525.
Traumatic Marginal Alopecia Due to Traction on Hair: Comparative Study of Alopecia Liminaris Frontalis of Sabouraud. O. G. Costa and M. A. Junqueira.—p. 527.

Treatment of Tinea with Ethyl Chloride.—Bograd states that treatment of lesions of the tinea group with ethyl chloride has given uniformly excellent results in the South Pacific. There are no contraindications to its use, even though the lesions may be covered by pustular eruptions due to secondary infections. The length of treatment depends solely on the age of the infection and not on the area it covers. All of it must be treated at one sitting. No other drug or dye should be used in conjunction with ethyl chloride. The ethyl chloride is sprayed on only until the temperature of the skin has been lowered sufficiently to bring out the white surface. Any tinea lesion, regardless of where it appears and whether it is acute or chronic, may be treated with ethyl chloride. The method is superior to that with any fungicide now used.

Fixed Eruption, Conjunctivitis and Fever from Sulfathiazole.—Director uses the term "fixed" in the sense that the dermatitis is confined to the same sites on recurring attacks. A sailor aged 34 entered the hospital on March 6 with a tender mass in the left groin of three weeks' duration and a penile sore of eight days' duration. Dark field examinations of material from the penile lesion did not reveal Treponema pallidum, and Kahn tests were negative. The Frei reaction was negative on March 9 and positive on March 29, 1943, establishing the diagnosis of lymphogranuloma venereum. The patient was kept in bed and given 3 Gm. of sulfathiazole daily from March 16 through March 20, when the mass in the left groin subsided. On March 17, after one day of sulfathiazole therapy, several dusky red, edematous macules appeared on both sides of the neck. On the next day the edema increased. On March 20 new macules broke out; others continued to appear until March 24. Small discrete red papules also appeared on the forehead, and both conjunctivas became diffusely injected. Sulfathiazole was discontinued on March 24, and the lesions began to fade by the next day; by March 26 they were almost completely gone, leaving a faint brown stain. On April 5 there was a recurrence of the swelling of the left inguinal lymph nodes. The patient was again given sulfathiazole from April 5 through April 7. At noon on April 7 there was a recurrence of the eruption on the neck and face. On April 19, prior to his discharge from the hospital, the patient was given 0.5 Gm. of sulfathiazole. Elevation of temperature accompanied by malaise appeared first after an interval of four hours and fifteen minutes and rose to 102.6 F. in two hours, remaining on this plateau for three hours and then subsiding to normal in six hours, a total febrile period of eleven hours. The conjunctival injection and dermatitis of the fixed sites on the neck and forehead appeared together. The injection disappeared completely in twenty-four hours. The same cutaneous sites previously affected flared into bright erythematous macules but did not become edematous. The dermatitis subsided completely in thirty-six hours. The patient did not recall ever having taken sulfathiazole before. The latent period of sensitization therefore could not have been more than twenty-four hours, since the original cutaneous lesions appeared one day after the first dose of the drug.

Arkansas Medical Society Journal, Fort Smith

40:103-116 (Nov.) 1943

The Doctor's Heart. J. H. Musser.—p. 103.

40:117-138 (Dec.) 1943

Cardiac Arrhythmias. O. C. Melson.—p. 117.

Cancer Research, Baltimore

3:809-928 (Dec.) 1943

Heterologous Transplantation of Embryonic Mammalian Tissues. H. S. N. Greene.—p. 809.
Influence of Egg White and Avidin Feeding on Tumor Growth. C. J. Kensler, C. Wadsworth, K. Sugiura, C. P. Rhoads, K. Dittmer and V. du Vigneaud.—p. 823.
Effect of Pyridoxine on Tumor Growth. B. E. Kline, H. P. Rusch, C. A. Baumann and P. S. Lavik.—p. 825.
Free Choice Dietary Study of Tumor Bearing Rats. W. M. Cahill, W. F. Dunning and A. H. Smith.—p. 830.
Vitamin A and the Toxic Action of Dibenzanthracene on Tissues. A. Goerner and M. Margaret Goerner.—p. 833.
Technic for Production of Hypothermia in Albino Rats. R. M. Hill, A. G. Ware and F. H. Schultz.—p. 839.
Effects of Lowered Body Temperatures on Methylcholanthrene Fibrosarcomas in Rats. R. M. Hill, E. K. Rutledge, A. G. Ware, F. H. Schultz and W. H. Livingston.—p. 841.
Determination of Carcinogenic Hydrocarbons in Animal Tissue: Two Condition Fluorometry. J. A. Miller and C. A. Baumann.—p. 849.
Sarcoma Producing Factor Extractable from Transplanted Rat Fibrosarcomas. P. M. Aptekman, Helen Dean King and Margaret Reed Lewis.—p. 856.
Estrogen Administration to Aged Female Monkeys with No Resultant Tumors. E. T. Engle, C. Krakower and C. D. Haagensen.—p. 858.
Rhabdomyosarcoma: Rat Tumor 92, Institute of Cancer Research, Columbia University, New York. W. H. Lewis.—p. 867.
Retention of Radioactive Phosphorus in Leukemic Patients. S. Warren.—p. 872.

Endocrinology, Springfield, Ill.

33:261-332 (Nov.) 1943

Bioassay of Adrenocorticotropic Hormone. Miriam E. Simpson, H. M. Evans and C. H. Li.—p. 261.
Factors Affecting Action of Antigonadotropic Sera in Immature Rats. W. H. McShan, H. R. Wolfe and R. K. Meyer.—p. 269.
Assay of Adrenal Cortical Extracts in Adrenalectomized Rats Exposed to Cold. A. Roos.—p. 276.
Studies of Zimmermann Reaction: Factors Affecting Color Intensity; Relation of Molecular Structure to Color Production. L. P. Hansen, A. Cantarow, A. E. Rakoff and K. E. Paschkis.—p. 282.
Color Measurement of Sexual Skin of Macacus Rhesus by Munsell System of Color Annotation. R. Cleveland, Susan Wilkes and Grace Sabotka.—p. 289.
Alloxan Diabetes in Dog. M. G. Goldner and G. Gomori.—p. 297.
Studies on Inactivation of Estradiol by Liver. A Cantarow, K. E. Paschkis, A. E. Rakoff and L. P. Hansen.—p. 309.
Effects of Estrone, Ascorbic Acid and Testosterone Propionate on Nitrogen Storage and Insulin Requirement in Dogs. O. H. Gaebler and S. M. Tarnowski.—p. 317.
Effects of Adrenal Cortical Compounds on Lactation. W. O. Nelson, R. Gaunt and Malvina Schweizer.—p. 325.

Journal of Experimental Medicine, New York

78:327-424 (Nov.) 1943

Pneumococcal Capsular Swelling Reaction, Studied with Aid of Electron Microscope. S. Mudd, F. Heinmets and T. F. Anderson. —p. 327.
Production of Scurvy-like Condition by Feeding of Compound Structurally Related to Ascorbic Acid. D. W. Woolley and L. O. Krampitz.—p. 333.
Yield of Rabies Virus in Chick Embryo. B. Sigurdsson.—p. 341.
Immunologic Response to Influenza Virus Infection as Measured by Complement Fixation Test: Relation of Complement Fixing Antigen to Virus Particle. W. F. Friedewald.—p. 347.
Kinetic Analysis of Renin-Angiotonin Pressor System and Standardization of Enzymes Renin and Angiotonase. A. A. Plentl and I. H. Page.—p. 367.
*Bactericidal Action of Propylene Glycol Vapor on Micro-Organisms Suspended in Air: II. Influence of Various Factors on Activity of Vapor. T. T. Puck, O. H. Robertson and H. M. Lemon.—p. 387.
Studies of Antigenic Differences Among Strains of Influenza A by Means of Red Cell Agglutination. G. K. Hirst.—p. 407.

Bactericidal Action of Propylene Glycol Vapor on Micro-Organisms in Air.—Puck and his associates state that pneumococci, hemolytic streptococci, staphylococci, Haemophilus influenzae and other organisms, as well as influenza virus, when sprayed into atmospheres containing propylene glycol vapors were killed so rapidly that no micro-organisms or virus could be recovered from the test chamber. Propylene and triethylene glycols are relatively nontoxic and in vapor form are odorless, tasteless and nonirritating to the respiratory mucosa. The authors studied factors which influence or limit the action of propylene glycol vapor. The killing process was more effective when both the total number of air borne droplets and the number of organisms in the bacterial suspension were small. A temperature below 80 F. and an atmospheric relative humidity between 45 and 70 per cent were found to constitute the most favorable conditions for the lethal action of the vapor. Equally strong bactericidal action was obtained when propylene glycol was dispersed in an 800 cubic foot room as occurred in chambers of 2 cubic foot capacity. Susceptibility to vapor action of bacteria resuspended in saliva was just as great as when broth was used as the suspending medium. Both partially and completely dehydrated bacteria succumbed to the effects of the vapor. When unsterile dust collected from inhabited rooms was dispersed into the air, little reduction of the natural microbic population contained in this material was observed. The minimum glycol concentration necessary for effective bactericidal action on various micro-organisms was as follows: Pneumococci were killed by amounts of propylene glycol as low as 1 Gm. in 20 million cc. of air; concentrations of 1 to 5 million to 1 to 10 million were required to produce the same degree of killing of streptococci and staphylococci.

Journal of Lab. and Clinical Medicine, St. Louis

28:1655-1774 (Nov.) 1943

Absorption and Excretion of Methylsalicylate Administered by Inunction. R. Beutner, B. Calesnick, E. Powell and L. Bortin.—p. 1655.
Systemic Allergic Reaction Induced by Yellow Fever Vaccine. H. Swartz.—p. 1663.
Pharmacology of Two Water Soluble Vitamin K-like Substances. J. J. Smith, A. C. Ivy and R. H. K. Foster.—p. 1667.
Effects of Vitamin B Deprivation on Spontaneous Activity of Rat. A. L. Bloomfield and M. L. Tainter.—p. 1680.
*Reaction of Leukemic Patients to Sulfonamides. E. L. Amidon. —p. 1691.
Effects of Various Intensities of Light on Certain Laboratory Animals. M. E. Maun and L. H. Domeier.—p. 1696.
Urease Activity of Proteus and Salmonella Organisms. W. W. Ferguson and A. E. Hook.—p. 1715.
Cephalin Flocculation Test in Jaundice. P. A. Kirschner and S. I. Glickman.—p. 1721.
Observations on Ingestion of Methyl Cellulose and Ethyl Cellulose by Rats. W. Deichmann and S. Witherup.—p. 1725.

Reaction of Leukemic Patients to Sulfonamides.—Livingston and Moore administered sulfapyridine to 6 patients with lymphatic leukemia and reported a definite change in the total number of lymphocytes in the peripheral blood. Amidon reports 4 cases of lymphatic leukemia in which he employed sulfonamides. The first patient reacted promptly to sulfanilamide and sulfathiazole by a drop in the total white cells followed by clinical improvement from a moribund state to that of comparative health. Contrary to prevailing teachings, the lymphocytes were decreased while the number of neurophils remained nearly constant. Because of the prolonged leukopenia it was thought best to discontinue the sulfonamides. Patient 2 showed the smallest hematologic change after the administration of sulfathiazole. He received only 6 Gm. over a four day period. The sudden vascular collapse after the administration of sulfathiazole is difficult to explain. The third patient, like the first, had the acute form of the disease and rallied from a nearly moribund state after sulfathiazole was given. The fourth patient was at the point of death on admission. He responded so well to sulfanilamide that he was able to be discharged from the hospital and was able to return to his job as janitor. On this admission he responded well to sulfathiazole but failed to show any change when sulfadiazine was given.

Journal of Urology, Baltimore

50:515-640 (Nov.) 1943

Experiences with Deming Nephropexy. A. I. Dodson.—p. 515.
Partial Resection of Kidney: Report of 13 Cases. C. P. Mathé. —p. 525.
*Renal Trauma and Its Relation to Hypertension. W. F. Braasch and G. W. Strom.—p. 543.
Lateral Nonrenal Retroperitoneal Neoplasms: Report of 2 Cases; Cortical Carcinoma of Adrenal Gland; Perineural Blastoma. L. Herman.—p. 550.
Crossed Renal Ectopia: Report of 2 Additional Cases. H. A. Zide and E. J. Maher.—p. 560.
Sarcoma of Kidney. W. Weisel, M. B. Dockerty and J. T. Priestley. —p. 564.
Experimental Use of Vitallium Tubes in Ureterointestinal Anastomosis. A. R. Stevens and J. W. Lord Jr.—p. 574.
Autopsy Twenty-Two Years After Cutaneous Ureterostomy for Tuberculosis. E. L. Keyes.—p. 580.
Late Results Following Transplantation of Ureters into Rectosigmoid. W. E. Lower.—p. 581.
Tumor of Testicle; Analysis of Results Obtained in 50 Patients Treated by X-Ray Therapy. G. G. Smith.—p. 585.
Benign Prostatic Obstruction in Young Adult. J. K. Ormond and R. B. Roth.—p. 590.
Intraprostatic Injection of Pitressin and Adrenalin in Control of Bleeding During Transurethral Resection: Preliminary Report. C. D. Creevy.—p. 593.
Retothel Sarcoma of Prostate Gland: Report of Case. J. D. Kirshbaum, H. S. Larkin and H. Culver.—p. 597.
*Orthostatic Albuminuria. C. L. Prince.—p. 608.
Intravenous Urokymography: Preliminary Report. B. Hughes. —p. 621.
Clinical Use of Sulfadiazine in Nonspecific Urinary Tract Infections: Study of 100 Cases. L. W. La Towsky.—p. 625.
Dawn of Genitourinary Surgery. A. Castiglioni.—p. 632.

Renal Trauma and Its Relation to Hypertension.—Braasch and Strom analyzed cases with clinical evidence of previous renal trauma in the records of the Mayo Clinic. There were 50 cases with a definite history of renal injury to a presumably normal kidney in which the blood pressure was recorded at some time during the patient's examination at the clinic. Nephrectomy was performed in 5 of these. Hypertension existed in 3 of these 5 cases. Following removal of the kidney, the blood pressure returned to normal. In the other 2 cases the preoperative blood pressure was normal. The small number of patients with renal injury who underwent nephrectomy corroborates the value of conservative treatment. In 38 of 45 cases in which no operation was indicated the blood pressure was normal subsequent to renal injury. In 6 cases the degree of hypertension was moderate and the hypertension either existed prior to renal injury or was explained by factors other than renal. In only 1 of these cases was renal trauma considered a possible factor in the production of hypertension. The changes in the renal tissue following injury usually do not cause hypertension. Hypertension due to renal trauma usually occurs in cases in which there is a secondary renal infection. This is in keeping with the fact that changes in renal tissues secondary to chronic infection have been noted in most renal lesions which are known to cause hypertension. There was 1 exception, a case of hypertension observed nine months after injury, in which an encapsulated hematoma was compressing a large portion of the kidney without infection. When hypertension exists following renal injury it may be difficult to determine whether the injury is an etiologic factor unless the blood pressure and other clinical records prior to injury are available.

Orthostatic Albuminuria.—Prince studied 5 young men who were rejected by medical examining boards because albumin was found in their urine. Four of the patients have since been accepted for active duty in the armed services after thorough examination proved that the albuminuria was orthostatic in

character. In the fifth case the albuminuria was found not to be orthostatic. A diagnosis of orthostatic albuminuria cannot be made immediately after a routine examination and a few urinalyses. Chronic glomerulonephritis in a latent stage, the condition most frequently confused with orthostatic albuminuria, must be ruled out. The author examines repeatedly for albumin the first morning specimen of urine voided immediately on rising. He considers normal intravenous phenolsulfonphthalein, dilution and concentration tests to be sufficient evidence of satisfactory renal function, although urea clearance and other renal function tests are frequently used as additional proof. A normal blood nonprotein nitrogen or urea nitrogen is essential. The importance of the intravenous urogram cannot be overemphasized. A normal intravenous urogram is absolutely essential before a diagnosis of orthostatic albuminuria can be made. The "lordotic" tests are most important and extremely helpful. Orthostatic albuminuria is quite harmless and usually disappears shortly after puberty. It has no effect on general health or longevity. Albuminuria of this type requires no treatment or curtailment of activity. There is no reason why young persons with orthostatic albuminuria should not be allowed to serve with the armed forces.

Medical Annals of District of Columbia, Washington

12:373-416 (Oct.) 1943

Electrocardiographic Changes Following Metrazol Convulsions: Effect of Prolonged Anoxemia. W. D. Claudy.—p. 373.
Common Skin Diseases of Children Seen in Private Practice. W. F. Burdick.—p. 383.
Demerol, a New Analgesic. C. S. White.—p. 388.
Functional Nervous Disorders in General Practice. J. R. Cavanagh.—p. 391.

Missouri State Medical Assn. Journal, St. Louis

40:339-362 (Nov.) 1943

Certain Considerations in Management of Small Intestinal Obstruction. C. J. Hunt.—p. 339.
Congenital Diaphragmatic Hernia: Report of Case. J. A. Ossman.—p. 341.
What Constitutes Adequate Examination? E. C. Funsch.—p. 343.
Industrial Hazards. W. M. Macon.—p. 344.
Continuous Caudal Anesthesia in Obstetrics. P. H. Lorhan.—p. 346.
Continuous Spinal Anesthesia. J. McNearney.—p. 348.

40:363-390 (Dec.) 1943

Blood Transfusions and Blood Substitutes. R. O. Muether.—p. 363.
Pulmonary Lesions of Sulfonamide. H. Pinkerton.—p. 364.
Coroner Plan and Medical Examiner System: Brief Comparative Survey of Statutory Provisions in England and Several American States. H. S. Breyfogle.—p. 366.

New England Journal of Medicine, Boston

229:667-700 (Oct. 28) 1943

Civilian Medical Defense and Its Adaptation to Peacetime Civil Life. A. W. Reggio.—p. 667.
*Treatment of Angina Pectoris: Summary of Ten Years' Objective Study. J. E. F. Riseman.—p. 670.
Laryngopyocele: Report of Case. O. A. Lothrop.—p. 681.
Pancreatic Insufficiency and Celiac Syndrome (concluded). S. Farber.—p. 682.

Angina Pectoris.—Riseman reports objective studies of the value of treatment in angina pectoris which have been carried out in a special clinic of the Beth Israel Hospital since 1933. To date sixty-eight different methods of chemical, physical and surgical therapy have been studied by clinical and objective procedures that permit unbiased comparison of effectiveness. It was found that 27 per cent of patients respond strikingly to practically all forms of therapy, 33 per cent respond to a moderate degree and 40 per cent usually fail to respond appreciably. This variation in therapeutic response should be taken into account in any study of the efficacy of treatment of angina pectoris. The clinical evaluation of the efficacy of therapy is extremely difficult and unreliable. Nothing short of complete or almost complete disappearance of attacks can be considered as a favorable response. Such periods may be spontaneous and not related to treatment. Of the sixty-eight methods of therapy evaluated, twenty were found to be of considerable value, twenty-two were of slight value and twenty-six were only of psychologic value. In prescribing medicinal therapy the drugs of choice are glyceryl trinitrate 1/400 grain (0.16 mg.) every hour, theobromine and sodium acetate 7½ grains (0.5 Gm.) four times daily, quinidine sulfate 3 to 5 grains (0.2 to 0.32 Gm.) four times daily, atropine sulfate 1/150 grain (0.4 mg.) four times daily, enteric coated potassium iodide 1 Gm. four times daily, cobra venom given intramuscularly and the sedatives. Few patients require surgery. Total thyroidectomy is of considerable value for selected patients. The paravertebral injection of alcohol gives symptomatic relief to others.

229:701-736 (Nov. 4) 1943

*Studies on the Destruction of Red Blood Cells: III. Mechanism and Complications of Hemoglobinuria in Patients with Thermal Burns: Spherocytosis and Increased Osmotic Fragility of Red Blood Cells. S. C. Shen and T. H. Ham, with the assistance of Eleanor M. Fleming.—p. 701.
Relation Between the Family Physician and War Industry. K. Pickard.—p. 714.
Pneumococcal Meningitis: Recovery Following Treatment with Sulfadiazine and Specific Antiserum: Report of Case. J. H. Young, C. Schlosberg, C. N. Gettes and S. A. Manconi.—p. 716.
Anemias of Pregnancy. R. A. Dillon.—p. 718.

Destruction of Red Blood Cells: Hemoglobinuria in Burns.—Shen and his co-worker's report observations on the blood and urine and on the kidney complications occurring in 14 patients with burns, of whom 10 showed hemoglobinuria. The authors report experimental studies on the effect of heating human red cells and on the effect of injecting heated canine red cells into the dog. Gross hemoglobinuria was observed in 9 patients and minimal hemoglobinuria in 2 among 40 patients with second and third degree thermal burns involving 15 to 65 per cent of the body area. The maximum excretion of hemoglobin occurred during the first twelve to twenty-four hours. The urine samples from patients with gross hemoglobinuria were scanty for one or two days, varied from black to red to light brown and were acid (p_H 4.5 to 5.8); they contained hemoglobin in solution, in precipitated form and in casts. The red and brown pigments were identified spectroscopically as oxyhemoglobin mixed with traces of methemoglobin. Eight cases showed hemoglobinemia. Oxyhemoglobin and methemalbumin were detected spectroscopically in the serum of 1 patient. Chronic azotemia occurred in spite of the reestablishment of adequate excretion of urine in 4 of 5 patients with hemoglobinuria who lived five days or longer. In 6 patients with hemoglobinuria examined post mortem the microscopic aspects of the kidneys were consistent with hemoglobinuria. Attempts to establish diuresis of alkaline urine should be made in all severely burned patients. No significant anemia developed during the first forty-eight hours in the patients with hemoglobinuria. If blood is rapidly heated to a temperature of from 51 to 65 C. (123 to 149 F.) there results fragmentation and the formation of spherocytes and microspherocytes, with increase in osmotic fragility and hemolysis of the erythrocytes. In patients with severe or moderately severe burns the red cells exhibited changes in morphology and osmotic fragility similar to those obtained by the injection into dogs of the animals' own erythrocytes heated in vitro to approximately 53 C. (127.4 F.). A significant number of erythrocytes may be destroyed by heat when burns are sustained, probably depending on the temperature attained by the blood, the duration of heating and the volume of blood subjected to these conditions.

New Orleans Medical and Surgical Journal

96:177-238 (Nov.) 1943

Pneumonia in Army. E. V. Allen and L. W. Baird.—p. 177.
*Sylvatic Plague: Probable Origin in United States, Distribution, Potentialities as Reservoir for Infections in Man. S. A. Trufant.—p. 184.
Common Diseases and Injuries of Spine. E. W. Ryerson.—p. 195.
Indications for Vitamin Therapy. Grace A. Goldsmith.—p. 199.
Unusual Infections in Newborn Caused by Staphylococcus Aureus. Ruth G. Aleman.—p. 207.
Warren Stone. R. G. Allen.—p. 213.

Sylvatic Plague.—Trufant presents a historical review of plague in the United States. According to the importation theory, infected rats carried the plague in ships from the Orient to San Francisco. The enzootic theory suggests that plague infection is enzootic in the Western United States, as it is in Asia and probably the steppes of Russia and in South Africa. Plague in wild rodents exists in at least twelve of the Western

states. Localized epizootics occur usually in the early spring. A vector is substantially indispensable to complete the propagation of plague among wild rodents. For the ground squirrel the vector is the flea Diamanus montanus. This flea not only harbored Pasteurella pestis but also conveyed it from squirrel to squirrel. These fleas are known to be capable of transmission of the infection after several months of "storage" and starvation. Very low temperature seems to increase the period of infectivity. So far spontaneous infections or reservoirs of plague have been established in over 31 rodents and in rabbits, the former including ground squirrels, marmots, chipmunks, wood rats, kangaroo rats and prairie dogs. Plague once established in a wild rodent population persists, but what the mechanism is has not been demonstrated. The author presents tables which list infected rodents and arthropods and the states in which they were found. Sylvatic plague as transferred to man in the United States was not pneumonic or extremely virulent. However, it is at least theoretically possible that plague might appear in epidemic proportions provided certain conditions as yet inadequately understood are conducive to an increased distribution of the infective agent.

North Carolina Medical Journal, Winston-Salem

4:457-496 (Nov.) 1943

Arteriosclerosis. W. Dock.—p. 457.
Recent Advances in Treatment of Hypertension. J. R. Williams Jr. —p. 460.
Hypertension Due to Unilateral Renal Disease: Report of Case. R. E. Brooks.—p. 463.
Combined Isometric and Stereoscopic Technic for Radiographic Examination of Obstetric Patient. C. T. Javert.—p. 465.
Typhoid Fever in Children. A. McBryde and F. Ledesma-Diaz. —p. 473.
Some Common Diseases of External Ear. A. J. Ellington.—p. 478.

Western J. Surg., Obst. & Gynecology, Portland, Ore.

51:419-462 (Nov.) 1943

*Experimental Evaluation of Succinylsulfathiazole and Phthalylsulfathiazole. A. C. Kirchhof, C. A. Racely, A. I. Thompson and N. A. David.—p. 419.
Pulmonary Embolism During Continuous Caudal Anesthesia: Report of a Maternal Death. A. W. Diddle and A. M. Hill.—p. 427.
Prolan A in Diagnosis of Teratoma Testis. T. G. McDougall and A. P. Grahma.—p. 432.
Preeclampsia. W. S. Morse.—p. 436.
New Type of Drainage in Gallbladder Field. R. C. Chaffin.—p. 440.
Pyelonephritis. G. Prewitt.—p. 450.

Experimental Evaluation of Succinylsulfathiazole and Phthalylsulfathiazole.—Kirchhof and his associates compared succinylsulfathiazole and phthalylsulfathiazole in studies on dogs. They found that total bacterial counts were not decreased consistently with either drug. No signs of toxicity were noted in any dog during or after medication. Maximum blood sulfathiazole concentrations were 1.3 mg. free and 1.6 mg. total per hundred cubic centimeters. Stool consistency was more normal in dogs receiving phthalylsulfathiazole than in those receiving succinylsulfathiazole. The authors emphatically disagree with Poth's suggestion of the prophylactic administration of succinylsulfathiazole to prevent bacillary dysentery in the armed forces who happen to be in insanitary surroundings. The authors think that definite clinical indications should be present before administration of any of the sulfonamides. Their observations show that even if the coliform count is greatly reduced the total count of viable bacteria remains high. Since no guaranty can be made for the nonpathogenicity of this group of bacteria, anticipation of a "sterile bowel" is wishful thinking. Surely no one should relax aseptic technics because of reliance on any of the drugs now available. If the surgeon feels that the hazards of toxic reactions or of establishing sensitivity are of less consequence than a reduced count of Endamoeba coli, he is justified in using this type of preoperative management.

Wisconsin Medical Journal, Madison

42:1113-1212 (Nov.) 1943

Administrative and Professional Problems of Medical Practice in the Hospital. A. M. Schwitalla.—p. 1139.
Professional Accounting. H. M. Coon.—p. 1148.
Hospitals and Selection of Medical Staffs. J. W. Holloway Jr.—p. 1151.
Interhospital Relations. W. A. Coventry.—p. 1154.
Problems Concerning Graduate Education and Employment of Nurses, Interns and Residents. W. A. O'Brien.—p. 1157.
Attendant Nursing. Katharine Shephard.—p. 1162.

FOREIGN

An asterisk (*) before a title indicates that the article is abstracted below. Single case reports and trials of new drugs are usually omitted.

Australian J. Exper. Biol. and M. Science, Adelaide

21:133-190 (Sept.) 1943. **Partial Index**

d-Fructo-Pyranose, A Sugar Unfermentable by Yeast. A. Gottschall. —p. 133.
Capillary Fat Embolism in Brains of Sheep, Pigs and Monkeys, with Especial Reference to Demyelination and Other Lesions in the White Matter. E. W. Hurst and Barbara Ternent Cooke.—p. 141.
Effect of Salicylates (*a*) On the Estimation of Thiamine by the Thiochrome Method, (*b*) On the Excretion of Thiamine. Joan B. Cleland.—p. 153.
Method for Colorimetric Determination of Oxyhemoglobin. H. F. Holden.—p. 169.
New Salmonella Type: Salmonella Adelaide. Nancy Atkinson.—p. 171.
Hyperiein and a Nonfluorescent Photosensitive Pigment from St. John's Wort (Hypericum Perforatum). R. C. Betty and V. M. Trikojus.—p. 175.
Pharmacology and Toxicity of Alstonia Alkaloids. Patricia Keogh and F. H. Shaw.—p. 183.
Some Observations on Excretion of Ketosteroids and Estrogens in Urine of Sheep. W. K. Whitten.—p. 187.

South African Medical Journal, Cape Town

17:279-294 (Sept. 25) 1943

*Treatment of Vomiting of Pregnancy by Drugs Which Stimulate Para-Sympathetic Nervous System. A. I. L. Turnbull.—p. 279.
Theories of Hearing from Morphologic Standpoint. J. A. Keen. —p. 283.
Sparganum from East African Native. B. de Meillon and R. B. Leech. —p. 289.
Expectation of Life and Expectation of Cancer in Union of South Africa. C. Pijper.—p. 290.
Uncertainties About Indentity of Adult Schistosomes. F. G. Cawston. —p. 291.
Further Note on Onyalai in Bechuanaland Protectorate. B. T. Squires. —p. 292.
Distribution of "Knopiespinnekop" (Latrodectus Indistinctus). R. H. N. Smithers.—p. 293.

Vomiting of Pregnancy.—According to Turnbull, carbohydrate starvation is one of the main factors in the causation of the vomiting of pregnancy. Hypertonus of the sympathetic nervous system tends to deplete the liver and muscle glycogen, and this might produce a state of carbohydrate starvation. A drug such as physostigmine sulfate, which stimulates the parasympathetic nervous system, should counteract a state of sympathicotonia. In order to investigate this hypothesis cases of vomiting of pregnancy were treated with physostigmine sulfate in conjunction with a diet rich in carbohydrates. Of 31 patients 29 recovered, 2 failed to respond to treatment and the pregnancy had to be terminated. In 4 cases the treatment was interrupted on several occasions; sometimes two or three days intervened between doses. In the remaining 25 cases the treatment, once begun, was carried out regularly with daily injections or oral administrations of physostigmine. Doses of 1/100 grain (0.00065 Gm.) should be given two or three times daily. If the patient is unable to take it by mouth it should be given hypodermically. Within fifteen minutes the patient should take a tablespoon of dextrose in a glass of water flavored with lemon or orange juice. The diet should be rich in carbohydrates and poor in fats. The author suggests that sympathicotonia plays a part also in eclampsia, hypertension and albuminuria and recommends further investigations of the influence of the autonomic nervous system on the toxemias of pregnancy.

Transactions Royal Soc. Trop. Med. and Hyg., London

37:67-168 (Sept.) 1943

Leptospirosis in Southern Nigeria: Case. J. I. Lesh and D. A. Cannon.—p. 89.
Fever of Dengue Group Occurring in West Africa. G. M. Findlay and R. W. Brookfield.—p. 95.
Differential Diagnosis of Infantile Beriberi. Lydia Fehly.—p. 111.
Some Observations on Study and Control of Yellow Fever in Africa with Particular Reference to Anglo-Egyptian Sudan. R. Kirk. —p. 125.
Hemoglobinuria Following Administration of Plasmoquine. W. N. Mann, with Note by S. Smith.—p. 151.
Sulfapyridine in Typhoid Fever. M. Rachmilewitz and K. Braun. —p. 157.
*Phenothiazine in Treatment of Human Intestinal Helminthic Infestations. M. Elliott.—p. 163.

Phenothiazine in Human Helminthic Infestation.—Observations among the natives of West Africa convinced Elliott that phenothiazine may be a potent destroyer of hookworms. He was also impressed by its action against Strongy-

loides stercoralis, parasites which up to this time have been very difficult to deal with in some patients. Kuitunen-Ekbaum in Canada studied its action against threadworms and found that it exercised a powerful destructive action. The author has given the drug to over 70 adult natives and has been impressed by its lack of unpleasant sequelae as compared with 50 control patients to whom santonin, oil of chenopodium or carbon tetrachloride had been given. He thinks that the American standard dose of 1 Gm. per 10 pounds (4.5 Kg.) of body weight is a safe level, because he experienced no intolerance with even twice this dose. To adults he gave over a period of four or five days a total dosage that varied between 20 and 30 Gm. Two 1 Gm. tablets are usually given three times a day. The tablets should be crushed. Of 36 patients with Ancylostoma duodenale 26 were cured, of 8 with Strongyloides stercoralis 5, of 15 with Ascaris lumbricoides 9, of 8 with Taenia saginata 5, of 8 with Trichuris trichiura 5 and of 9 with Endamoeba histolytica 6.

Revista Clínica Española, Madrid

9:365-442 (June 30) 1943

*Erythroblastosis Neonatorum. M. María de Mendizábal, E. de Amilibia and J. S. Harguindey.—p. 366.
Cervicobrachial Syndrome Due to Cervical Rib. J. P. Cornago and J. A. Tercedor Avilés.—p. 401.

Erythroblastosis in Newborn Infants.—Mendizábal and his collaborators observed 2 mothers of 10 newborn infants with erythroblastosis fetalis. One mother had four pregnancies: (1) Her first infant was normal and is 6 years of age; (2) there was a set of twins with acute pallor who died within six days; (3) an infant with jaundice and subcutaneous hemorrhages died within three days, and (4) an infant with acute hydrops died a few minutes after birth. The blood of the mother, her husband, the child and the fetus are of the same blood group. However, the blood of the mother agglutinated that of the husband, of the child and of the fetus. The second mother, 34 years old, had three abortions and four pregnancies with deliveries of living infants. The first two infants died within three days of acute jaundice; the third infant died within three hours after birth of acute anemia; the set of twins from the fourth pregnancy died of acute jaundice within four days. The blood of the mother agglutinated the blood of the husband and of the last fetuses. A large number of erythroblasts were observed in the blood of the placenta of 1 of the fetuses and in the blood of the liver of another. All fetuses were male. Neither couple had syphilis. The blood of any of the 2 women did not agglutinate that of the other. It did not agglutinate with control A_1MN blood serum. The blood serum of both women caused agglutination in blood of the largest number of persons of the O group blood. The results suggest the presence of a typical anti Rh agglutinins in the blood of both women and the pathogenic role of isoimmunization in erythroblastosis fetalis. One of the women had progesterone in large doses during pregnancy of the twins who were born with jaundice.

Revista Médica de Chile, Santiago

71:829-944 (Sept.) 1943. **Partial Index**

Hemolytic and Nonhemolytic Familial Jaundice. H. Ducci, R. Etcheverry, A. Nijamkin and J. Zañartu.—p. 837.
Mammary Carcinoma. A. Rahausen and C. Sayago.—p. 853.
*Prothrombinemia and Vitamin K in 106 Patients with Chronic Pulmonary Tuberculosis. J. Motlis and J. Guash.—p. 871.

Prothrombin in Pulmonary Tuberculosis.—The prothrombin time was determined according to Quick's technic in 106 cases of chronic pulmonary tuberculosis observed by Motlis and Guash. In 30 per cent of the cases there was a significant decrease of the prothrombin level in the blood. No relationship could be established between the presence of low prothrombin levels and the occurrence of hemoptyses, the stage of development of the disease, the presence of tuberculous intestinal lesions or the microscopic appearance of the liver. In the cases presenting hemoptyses, with or without low prothrombin levels, the intravenous administration of vitamin K had no effect on the severity, frequency or continuation of the hemoptyses. Severe and extensive pulmonary lesions were, as a rule, associated with the most noticeable decrease in prothrombin level, and Motlis and Guash point out that in this sense the test may have some prognostic value.

Deutsche Zeitschrift für Chirurgie, Berlin

255:667-816 (July 20) 1942

*Pathologic Anatomic Observations on Surgically Removed Sympathetic Cervical Ganglions in Bronchial Asthma. E. Hagen.—p. 667.
Clinical Studies on Postoperative Pulmonary Suppurations. G. Guerreri d'Antona.—p. 700.
Postoperative Pulmonary Complications. A Fehr, C. Molo and O. Walther.—p. 732.
Giant Echinococcus Cyst of Kidney with Complete Disappearance of Renal Parenchyma. V. Lazarević.—p. 747.
Myiasis Linguae—Two Maggots of Wohlfahrtia Magnifica in Papillae Circumvallatae of a Previously Healthy Tongue. N. Guntscheff.—p. 751.
Closure of Solitary Pulmonary Cyst by Muscular Plastics. O. Diebold.—p. 756.
*Treatment of Niche-Forming Gastric Ulcer and Relations to Carcinoma. M. Siebner.—p. 764.
Bone Transplantation Particularly with Os Purum and Os Novum. W. Stark.—p. 776.

Pathologic Anatomy of Sympathetic Cervical Ganglions Removed for Bronchial Asthma.—Hagen reports observations on surgically removed cervical ganglions of 7 patients with bronchial asthma. The formaldehyde fixed frozen sections were prepared according to the silver method of Bielschowsky-Gros. The author stresses the importance of this silver stain, pointing out that a large number of pathologic processes in ganglion cells can be visualized only by this method. The report is illustrated with excellent drawings. Detailed descriptions are given of the microscopic changes in the ganglion cells, the nerve fibers and the interstitial tissues. The majority of the ganglion cells show pathologic changes. They frequently exhibit a disharmony of processes with hypertrophic glomerulus-like ball formation. Vacuolation and granular degeneration are often seen in the body of the ganglion and in the processes. The appearance of spherical terminal formations in the processes is a sign of pathologic irritation. Accumulation of pigment as well as pathologic nuclear changes can be observed in the ganglion cells. The increased appearance of multinuclear ganglion cells and the pathologic changes observable in them represent another degenerative process in bronchial asthma. The hyperplasia of finest nerve fibers near diseased ganglion cells probably is a manifestation of pathologic growth and regeneration. Although the majority of ganglion cells show pathologic changes, the nerve fibers and the connecting branches emerging from the ganglion show no degenerative changes in the axis cylinders. Acute inflammatory processes are seen repeatedly in the interstitial cells. The microscopic changes indicate not only that bronchial asthma is a "functional" disorder but that serious organic changes exist in the sympathetic nervous system. It is possible that the changes in the sympathetic cervical ganglions are the cause of the secondary symptoms of asthma. The presence of multinuclear degenerated ganglion cells in the lower cervical ganglion suggests that asthma is possibly preceded by disturbances in the development of the sympathetic system which are decisive in the constitution of the asthmatic patient. The clinical symptoms of such patients with their allergic-hyperergic reactivity might result from a disturbance in the neuroendocrine system, and the treatment of asthma therefore should be directed toward this system.

Treatment of Niche-Forming Gastric Ulcer.—Siebner states that whereas the niche-forming ulcers require surgical treatment only in the case of rupture or in the presence of signs of threatening perforation, the chronic callous gastric ulcers with giant niches always require operation because medical treatment is often ineffective, because of the refractoriness and severity of the symptoms and because of repeated hemorrhages and increasing cachexia. Another reason for surgical treatment is the danger of malignant degeneration of the ulcer. This serious complication is best demonstrated by repeated roentgen examination. If the niche does not become smaller after from two to four weeks of ulcer treatment and if there are changes suggesting a malignant process in the mucosa adjacent to the ulcer, resection is to be carried out. Histories of several patients with giant niches suggest that diagnostic mistakes cannot always be avoided in spite of careful investigation of the early history, of the clinical and roentgenologic studies and even of careful macroscopic inspection of the resected specimen. Microscopic examination is necessary for an exact diagnosis.

Book Notices

Nervousness, Indigestion, and Pain. By Walter C. Alvarez, M.D., Professor of Medicine, University of Minnesota (Mayo Foundation), Rochester, Minnesota. Cloth. Price, $3. Pp. 488. New York & London: Paul B. Hoeber, Inc., 1943.

"This is a different sort of book—one which deals more with sick unhappy persons than with their diseases, more with symptoms and their meaning than with disease entities, more with the handling of patients than with the giving of medicines, and more with the puzzling, poorly understood and poorly described abdominal discomforts and indigestions than with the well known organic diseases such as ulcer, cholecystitis and cancer."

This opening statement from the preface describes an interesting and valuable distillation from the experience of an intelligent, humane and seasoned practitioner who has taken the time and made the effort to know his patients as human beings and thereby has found an important relationship between the emotional life and illness. Not only is the material different from the usual medical book but the chatty, informal manner in which it is written is a pleasant relief from the orthodox medical treatise. Even the chapter titles are different: "Help in Sizing Up the Patient," "Telling the Truth to Patients," "Handling the Nervous Patients" are examples. One of the most valuable chapters presents many helpful points in history taking, showing its importance in making a diagnosis. Equally emphasized throughout the book are errors which arise from too great a dependence on laboratory reports to the exclusion of good clinical judgment.

Although written from the general medical point of view and of interest to all physicians, the book deals chiefly with the three main complaints that gastroenterologists commonly find associated together—nervousness, indigestion and pain. Thus it will make its appeal particularly to specialists in this field. For them Dr. Alvarez has some very good advice: "Every gastroenterologist who hopes to be worthy of the name and would like to keep from making one serious blunder after another should be learning all he can about the psychiatry of the apparently sane. Like me, he may not have wanted to get into this field of medicine, but he cannot stay out of it and be a safe internist." If the experience of civil practice does not amply confirm this statement the medical experience of the present war surely does. Witness the emphasis on gastrointestinal disorders and their relation to the emotions and one must realize that, just as the first world war gave great impetus to the development of scientific psychiatry, so the present war is seeing its final integration into general medicine, in other words, psychosomatic medicine. Dr. Alvarez has furnished his fellow gastroenterologists with a fine introduction to this highly important subject. It must be emphasized, however, that it should be looked on only as an introduction because there is nothing to suggest that psychopathology and differential diagnosis based on personality structure must be understood in relation to disorders of emotional origin, just as tissue pathology serves as the background for an understanding of structural disease. Psychopathology bears the same relation to "nervousness" that histopathology does to organic disease. Indeed, if one was to find any fault with this interesting book it would be that Dr. Alvarez lumps all nervousness together and when he gets into difficulties he calls on all ill defined concept, "constitutional inadequacy," to suggest that further help is impossible. The concept of "constitutional inadequacy" is confused with psychoneurosis, and this reviewer questions whether such patients must resign themselves to their fate.

Like a wise and experienced gross pathologist who makes shrewd guesses from the gross appearance of diseased organs, so may the experienced clinician make shrewd guesses from gross distortions of the personality. But he is in a much happier position if he knows the underlying structure of the personality. Such knowledge is now available, and gradually our medical schools are teaching it.

More important, however, than this knowledge is Dr. Alvarez's sympathy and broad human understanding, his liking for people and his remarkable gift for understanding their troubles. If we would have to choose between these qualities and a knowledge of scientific psychopathology in trying to help our patients, by all means let us have the former. As much of these qualities as can be put in a book are in this one. It can be read with profit and it will certainly be read with pleasure.

First-Aid Training: A Study and Practice Book. By Morris Fishbein, M.D., Editor, Hygeia, the Health Magazine, Chicago, and Leslie W. Irwin, Ph.D., Director of Health and Physical Education, the Laboratory Schools, University of Chicago. Paper. Price, 80 cents. Pp. 216, with 57 illustrations. Chicago: Lyons & Carnahan, 1943.

This is a workbook intended for use in secondary schools. There are fourteen units, plus unit and review tests and lists of first aid supplies. There is a pronunciation word list. The units deal, in the order named, with the importance of knowing first aid; the body machine; shock, first aid for wounds; bleeding; asphyxia; internal poisoning; bandaging; injuries from heat and cold; puncture, gun and powder wounds; animal and insect and poisonous snake wounds; fractures, dislocations and sprains; everyday problems in first aid; first aid for the unconscious and emergency transportation of the injured. Each unit includes a factual presentation, well illustrated, a list of assigned activities and a practical problem, with a review section of "things to remember." The format is ingenious. It is bound in paper with pages perforated so that they may be torn out for handing in to be graded. The pages are also punched to fit a standard two ring binder so that they may be assembled in a binder. Or the binding may be detached from the whole book and all of it may be kept in a ring binder. This should be a very useful instrument wherever first aid is being taught on a practical basis.

Addendum to the Chemistry of the Amino Acids and Proteins Inclusive of Some of the Advances Since 1937. Edited by Carl L. A. Schmidt, M.S., Ph.D., Professor of Biochemistry and Dean of the College of Pharmacy, University of California, Berkeley. Cloth. Price, $5. Pp. 1035-1290, with illustrations. Springfield, Illinois, & Baltimore: Charles C Thomas, 1943.

In a field which is developing as rapidly as that of protein chemistry, a period of five years may bring with it many significant advances. Instead of completely revising the book "The Chemistry of the Amino Acids and Proteins," published in 1938, the editor and publisher of that work have seen fit to bring the subject matter up to date by the publication of this addendum. The chapter headings in this volume are the same as the chapter headings in the original work, and the text has been written in practically all cases by the original authors. Its pagination continues that of the first edition, and this addendum also appears as a supplement in the second edition of "The Chemistry of the Amino Acids and Proteins." Some chapters have been extended much more than others, as required by the amount of new material available. While it is questionable whether such a method of revision is completely satisfactory, this addendum is sure to be welcomed by all students of the subject, and especially by those who possess the original volume. For new readers interested in obtaining a comprehensive view of the whole field of modern protein chemistry, this book, when used in conjunction with the original work, still remains the best available source of information in this highly important field.

The Ear, Nose and Throat in the Services. By R. Scott Stevenson, M.D., F.R.C.S., Major R. A. M. C., Otologist to a Military Hospital. Oxford War Manuals. General Editor: The Rt. Hon. Lord Horder, G.C.V.O. Cloth. Price, $1.50; 5s. Pp. 116, with 16 illustrations. New York & London: Oxford University Press, 1943.

The author intended this manual as an aid for the average medical officer in the various military services as well as for those in civil life who may be called on to care for war casualties on the home front. Although the well trained otologist might read this work with no more than casual interest, it would if carefully studied be of great use for the others mentioned. The author writes well; he tries to discuss only what is essential, and he has adorned his little manual with drawings made presumably by himself. The size, furthermore, recommends it; fitting the coat pocket easily, it can be read in moments snatched from duty.

Queries and Minor Notes

THE ANSWERS HERE PUBLISHED HAVE BEEN PREPARED BY COMPETENT AUTHORITIES. THEY DO NOT, HOWEVER, REPRESENT THE OPINIONS OF ANY OFFICIAL BODIES UNLESS SPECIFICALLY STATED IN THE REPLY. ANONYMOUS COMMUNICATIONS AND QUERIES ON POSTAL CARDS WILL NOT BE NOTICED. EVERY LETTER MUST CONTAIN THE WRITER'S NAME AND ADDRESS, BUT THESE WILL BE OMITTED ON REQUEST.

PHYSICAL STRAIN AND CORONARY OCCLUSION

To the Editor:—What is the best opinion on the relation of strain in causing a coronary occlusion in a man aged 54 without previous history of heart disease? What do the records show in decisions made in compensation courts concerning the relation of coronary occlusion to stress? This coronary attack came on right after the man had lifted numerous shovels of sawdust weighing 35 pounds.

Bennie Mecklin, M.D., Watertown, N. Y.

ANSWER.—The balance of opinion concerning the direct relationship of physical strain to coronary thrombosis with myocardial infarction is that in the vast majority of cases there is no such relationship. This will probably amount to at least 95 per cent of the cases, but there are infrequent, clearcut instances in which excessive physical strain is accompanied or immediately followed by typical coronary occlusion in persons who may or may not have had angina pectoris previously. It is quite likely that an actual hemorrhagic lesion or some such development is precipitated in the coronary artery wall by the strain and that this in turn leads to the coronary occlusion.

Prof. Hubert Smith of the Harvard Law School has recently added the following statement to these comments:

The relation of coronary thrombosis to stress or unusual exertion is recognized by workmen's compensation commissions and courts to be a problem of scientific proof. Under compensation law preexisting heart disease does not bar compensation if an unusual exertion, not characteristic of the ordinary performance of that job, leads to cardiac injury or death. Such unusual exertions are considered to be "accidental injuries sustained in the course and scope of employment." Causal connection is not at present being proved with any great degree of scientific acumen. The compensation laws of a few states authorize the commissioners to order postmortem examinations, but such clauses are but rarely invoked, and the proof now rests principally on the "post hoc, ergo propter hoc" basis, causal inference being drawn from speedy effects following overexertion.

LOSS OF HAIR AND SULFONAMIDE THERAPY

To the Editor:—About two months ago I treated two women for peritonsillar abscesses with sulfathiazole. The total dosage in each particular case did not exceed 20 grains (1.3 Gm.) over a period of seven to eight days. Now these women have been complaining that they have been losing their hair, the onset of loss beginning about a week after the stopping of the sulfonamide. I cannot find anything wrong with the scalp. Has the loss of hair ever been reported as a complication of the use of sulfathiazole? Have you any suggestions as to the cause of this condition?

James A. Rooney, M.D., Newport News, Va.

ANSWER.—The loss of hair has not been reported as a complication of sulfathiazole. It is exceedingly doubtful that two women with the same disease, and given only 20 grains of sulfathiazole during a period of a week, should have a loss of hair because of the sulfonamide therapy. No mention is made about other complications being due to the sulfonamide, so the assumption is that none were present. It is not unlikely that the loss of hair might be due to the infection. It is well known that severe infections, particularly typhoid, may be associated with extensive but temporary loss of hair.

PREGNANCY AND SYPHILIS

To the Editor:—A woman received adequate antisyphilitic treatment, following which a negative Kahn reaction was reported. While carrying one child she continued receiving arsenicals and heavy metals. The child was born with a negative Kahn reaction. The mother is again pregnant and still has a negative blood reaction for syphilis. Should she receive more treatment while pregnant?

M.D., Michigan.

ANSWER.—Nothing is said as to how long the patient has had her syphilis. While she has a negative Kahn reaction, this does not mean that she is biologically cured. She may still have organisms in her system and on that account the Surgeon General's Cooperative Clinical Group has advised that all pregnant women with syphilis receive treatment throughout their pregnancy, emphasis being laid on arsenical therapy at the beginning, a short course of bismuth and arsenical therapy (mapharsen) at the end.

VALUE OF WIDAL TEST IN DIAGNOSIS OF TYPHOID

To the Editor:—During the past year there were several cases of typhoid in the community and the question of the diagnostic significance of the Widal test arose. In July 3 persons became ill almost simultaneously. Patients A and B were proved by blood and stool cultures to have typhoid. Patient C was ill for three weeks with temperatures ranging from 100 to 102 F. The only complaints were of general malaise and severe headache and backache. The pulse rate paralleled the degree of fever without significant bradycardia. There was a transitory macular rash during the first week of the disease, and the spleen became palpable during the second week. Otherwise physical examination was not helpful. During the first week of the disease the white blood cell count was 4,950 with 66 per cent polymorphonuclears, 31 per cent lymphocytes, 2 per cent monocytes and 1 per cent eosinophils. The blood count rose to 9,250 with 62 per cent lymphocytes during the second week and to 10,200 with 66 per cent lymphocytes during the third week of the disease. On the ninth day of the disease a Widal test was negative and blood culture was negative. On the fifteenth day the Widal became positive in a dilution of 1:160 and blood culture was again negative. On the thirtieth day the titer of the Widal had fallen to 1:40. Stools and urine were repeatedly negative throughout the course of the disease. Since the 3 cases developed in the same locality, case C was assumed to be typhoid despite the lack of bacteriologic confirmation. However, the epidemiologist from the state department of health stated that the Widal test in case C was of absolutely no significance unless a positive culture from the blood, urine or stools was obtained. Patient D, a laboratory technician who had been in contact with all 3 of the patients, had been given prophylactic inoculations for typhoid in the summer of 1942. During the first part of October 1943 she became ill with general symptoms of malaise, headache and bowel disturbance. Her temperature never rose above 101 F. although a low grade fever has now persisted for three weeks. Physical examination has revealed none of the confirmatory signs of typhoid, nor any others of significance other than a mild pharyngitis early in the disease. The blood count on the seventh day of the disease showed 16,000 white blood cells and 72 per cent polymorphonuclears. On the eighth day a Widal test was positive in a dilution of 1:640. This was confirmed by a positive Widal in the same dilution the following week. Agglutinations for Brucella abortus and paratyphoid were negative. Blood, stool and urine cultures have all been repeatedly negative. I would appreciate an opinion as to the significance of the positive Widal reactions in cases C and D.

M.D., Michigan.

ANSWER.—While it is true that in general a positive diagnosis of typhoid cannot be proved without cultivation of the organisms from the blood, a negative blood culture or even more than one negative does not exclude the diagnosis. A single positive preceded or followed by one or more negative culture is not a rare occurrence, illustrating the limitation of the method. Positive cultures from urine or stools, while of course usually highly significant, might indicate only a carrier condition, while a negative culture may mean that the time in the course of the disease was not opportune or that an unsuitable culture medium was selected. The significance of the Widal reaction in the cases referred to may well be the subject of difference of opinion, but the following would receive wide acceptance:

In case C, mention is not made of previous typhoid vaccination, which might obscure the situation; therefore the appearance of typhoid agglutinins might well be accepted as strongly suggestive of a diagnosis of typhoid.

In case D the prior typhoid vaccination might account for the presence of agglutinins; hardly likely, however, in dilutions as high as indicated, unless a nonspecific elevation had occurred. Even in this case unless another diagnosis could be substantiated the evidence would be considered as pointing strongly to typhoid, especially as typhoid is reported often to run an atypical course in previously vaccinated persons.

It might have been possible to derive more information had data been available on O and H agglutinins, especially in case D.

In the absence of data establishing another diagnosis, that of typhoid is highly probable in case C and a strong possibility in case D.

CONVALESCENT SERUM AND Rh FACTOR—Rh FACTOR IN HORSE AND RABBIT

To the Editor:—Is it possible to transmit the Rh factor in convalescent human serum? Has the Rh factor been found in the horse or rabbit?

Philip Leavitt, M.D., Brooklyn.

ANSWER.—The term Rh factor is properly applied only to the antigen or agglutinogen Rh in the erythrocytes. Accordingly, it would not be possible to transmit the Rh factor in convalescent human serum. Perhaps the inquirer when mentioning the Rh factor intends to refer to the isoantibody anti Rh, present in the serum of some persons sensitized to the factor. Since only 1 out of 7 persons are Rh negative and of these only approximately 1 out of 50 are easily sensitized, it is evident that only 1 among about 350 persons would be readily sensitized against the Rh factor. When, in addition, one considers that the only methods of sensitization known are by the repeated transfusions of Rh positive blood or by pregnancy with an Rh positive fetus, it is evident that the likelihood that donors of blood for the preparation of convalescent serum will have anti Rh isoanti-

bodies in their serum is extremely remote indeed. When one also bears in mind that usually pooled convalescent serum is used and the doses of such serum are small, it becomes obvious that the possible danger from this source is virtually nonexistent.

Thus far, Rh agglutinogens and natural anti Rh antibodies have not been demonstrated in the horse or rabbit. Immune Rh antibodies have been produced in the rabbit by the injection of rhesus monkey blood and human Rh positive blood, but similar experiments have not yet been tried in the horse.

POISONOUS FISH OF SOUTH PACIFIC

To the Editor:—As there are many varieties of fish in the South Pacific area, some of which are poisonous, it would be of assistance to us to know which of the fish are poisonous and when they are poisonous. Some of the local named fish are Tazar, Carauque, Ferroquet, and sardines which are considered not fit to eat. Rouget, Bossy and Bec de Caune are considered good to eat. It would be appreciated if we could have the names, especially common names, of the fish to be avoided.

Major, M. C., A. U. S.

ANSWER.—The great majority of tropical fishes are edible, although they vary greatly in palatability. They should be consumed when fresh, as they deteriorate rapidly. Occasionally even edible fishes are unwholesome because of feeding on certain kinds of food. The flesh of a few species is definitely poisonous, and some others are suspected of poisonous qualities. The safest guide is provided by the eating habits of the natives. Vernacular names are not of much value, as they change with the different localities. When known, the Hawaiian (H) and Samoan (S) names of the principal suspected groups are given in the following list:

Globefishes and Puffers.—Makimaki (H); Sue (S). Bodies balloon-like, capable of distention with air when taken from water; skin either smooth, like sandpaper or covered with erectile spines; teeth fused like a parrot's beak; flesh poisonous.

Filefishes.—Oili (H). Bodies laterally compressed; skin leathery or like sandpaper; a single, long, erectile spine on back; some species poisonous.

Triggerfishes.—Humuhumu (H); Sumu (S). Bodies diamond shaped viewed from the side; three erectile spines on back (graduated in length); usually a strong spine at lowest point of belly; often highly colored; occasionally eaten but considered unsafe.

Red Snapper.—Mumea (S). Shaped like our bass, but with a pointed nose; mouth with prominent canine teeth; dark red; grows to 2 feet in length; reported extremely dangerous.

Scorpions.—Omakaha (H); Nofu, Sausaulele (S). Resemble our sculpins, often with winglike pectoral fins; head bony with numerous blunt spines; row of sharp spines on back, which are provided with venom and are capable of inflicting a serious sting. Flesh is wholesome after spines and venom sacs are removed.

Poison Toadfish.—Nofu (S). Resembles a lump of lava or coral; slow swimming; hides among rocks on bottom; venom on spines deadly but flesh wholesome if spines and poison sacs are removed. Extremely dangerous to bathers.

Reference:

"Poisonous and Harmful Fishes," Bulletin 159, Council of Scientific and Industrial Research, Commonwealth of Australia. Pamphlet, 28 pages, pictures (some colored). Melbourne, 1943.

GAS IN VAGINA AND TRICHOMONAS VAGINALIS

To the Editor:—I should like to know the cause of the formation of gas in the vagina and its treatment. Lieutenant Commander (MC), U.S.N.R.

ANSWER.—Practically the only free gas that can be found in the vagina is that which escapes into the vagina from the rectum through a rectovaginal fistula and that which is expelled into the vagina from the uterus in the rare cases of physometra. Aside from these instances, gas in the the vagina nearly always exists in the form of foam. The latter condition is associated with Trichomonas vaginalis vaginitis. There is an excellent review of this subject in THE JOURNAL, Oct. 23, 1943, page 481.

There are a large number of ways of treating Trichomonas vaginalis vaginitis, and almost any type of therapy will quickly relieve the patient of her annoying discharge and irritation. However, recurrences are common regardless of the type of treatment. One of the simplest forms of treatment is as follows: The vulva, vagina and anal region are cleaned with tincture of green soap, and washed with tap water. The vagina and vulva are then thoroughly dried. Powder is blown into the vagina and on the cervix by means of a special blower, and some of the powder is also deposited on the vulva. Among the satisfactory powders are aldarsone, floraquin and picric acid. This treatment is carried out every second day for three or four times. If the cervix is eroded it should be cauterized. Vinegar or lactic acid douches are helpful.

CAFÉ AU LAIT SPOTS AND ALBRIGHT SYNDROME

To the Editor:—Will you please inform me as to the significance of "café au lait spots" and their relation to Albright's disease, a disease entity I am unable to find in the few textbooks that are available.

Lieutenant (MC), U.S.N.R.

ANSWER.—"Café au lait spots" are simply areas of brownish pigmentation within the skin. They may be single, discrete or disseminated, small or large, raised or flat. They are thought to be a result of some abnormal development of peripheral nerves and are frequently spoken of as cutaneous flat neurofibromas. Albright described a peculiar group of symptoms or findings which have become known as the "Albright syndrome." This is characterized by osteitis fibrosa, disseminating areas of pigmentation and, at least in the female, precocious sexual development. It was Albright's opinion that the condition represents an abnormal development within the central nervous system and is not primarily due to endocrine dysfunction. Braid has reported cases and expressed the opinion that this entire syndrome might be the result of icterus gravis neonatorum. His arguments do not seem to be borne out by the rather large series of cases studied by Albright.

References:

Albright, Fuller; Butler, Allan M.; Hampton, Aubrey V., and Smith, Patricia: Syndrome Characterized by Osteitis Fibrosa Disseminata, Areas of Pigmentation and Endocrine Dysfunction, with Precocious Puberty in Females, *New England J. Med.* **216**: 727 (April 29) 1937.

Albright, Fuller; Scoville, Beecher, and Sulkowitch, H. W.: Syndrome Characterized by Osteitis, Fibrosa Disseminata, Areas of Pigmentation and a Gonadal Dysfunction, *Endocrinology* **22**: 411 (April) 1938.

Braid, Frances: Osseous Dystrophy Following Icterus Gravis Neonatorum: Generalized Osteitis Fibrosa with Areas of Pigmentation of the Skin and Precocious Puberty in the Female, *Arch. Dis. Childhood* **14**: 181 (Sept.) 1939.

TIME OF INSULIN ADMINISTRATION IN RELATION TO MEALS

To the Editor:—Will you please tell me at what time, with relation to meals, insulin should be taken in moderate or mild cases of diabetes? Why is it advised many times to take it fifteen or twenty minutes before meals when the food would not get into the system until at least one or two hours after meals? Henry J. Winkler, M.D., L'Anse, Mich.

ANSWER.—In diabetic patients the absorption of both regular and crystalline insulin from the site of injection is most rapid during the first hour after injection, and thereafter the rate of absorption is much slower but continues for at least five hours. Similarly the sugar in the blood in the diabetic rises rapidly during the first thirty to sixty minutes after food and may not fall during the next few hours unless the action of insulin brings about a decline in the blood sugar. To secure the maximum benefit from insulin, its injection one-half hour before a meal makes active a larger part of the injected dose during the period of rapid rise in blood sugar than would be true if the insulin injection took place at a later time.

With protamine zinc insulin the time of injection is of less importance. Protamine zinc insulin acts so slowly that a difference of one-half hour counts little. On the other hand, if crystalline insulin and protamine zinc insulin are given separately or together before the meal, it is most desirable that food be taken within about fifteen minutes of the injection. The reason for this is that a patient under the influence of protamine zinc insulin tends to have the blood sugar well down to normal in the morning before breakfast, and a few additional units of a quick acting insulin at that time might depress it still more and a reaction might result.

Rules for giving insulin are not absolute. One must expect that changes will take place both in the absorption of food and in the absorption of insulin.

EDEMA OF ARM AFTER TYPHOID VACCINATION

To the Editor:—Recently in a fairly large series of injections of a booster dose of typhoid vaccine (U. S. Army 0.5 cc.) given in the usual manner I observed one soldier who developed an edema of the corresponding upper extremity. This reaction came on within one hour and extended from the finger tips up to the site of injection (deltoid region). The edema was fairly firm, pitted slightly, was not tender, was limited to the dorsal aspect and did not show any color change. There was no complaint of pain. The edema was of moderate degree. No therapy was advised. When observed the next day, about twenty hours after the injection, the extremity looked normal and there were no traces of edema anywhere. The injection site was normal in appearance. There was no general reaction.

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